The Australian and New Zealand Journal of Surgery

OCTOBER, 1941.

TABLE OF CONTENTS.

[The Whole of the Literary Matter in THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY is Copyright.]

ORIGINAL ARTICLES—

		PAGE
Indications for Opening the Common Bile Duct	*T. E. V. Hurley*	75
A Review of the Results of Suture of the Common Bile-Duct after Exploration	*B. T. Edye*	78
Historical Transfusions	*E. L. Cooper*	84
Traumatic Obstruction of the Axillary Vein (Primary Thrombosis of the Axillary Vein), with a Report of Two Cases	*R. O'Regan*	88
A Malformation of the Hind End of the Body	*S. Sunderland*	97
Diverticulosis and Diverticulitis	*H. R. G. Poate*	110

SURGICAL TECHNIQUE—

Instrumental Methods of Tying Knots in Inaccessible Regions	*A. C. McEachern*	123

CASE REPORTS—

A Case of Congenital Ectopic Anus Treated by Operation	*D. Robb*	125
Two Recent Cases of Slipped Femoral Epiphysis Treated by Pin Traction	*H. K. Christie and B. T. Hooper*	127
Benign Papilloma of the Ureter	*G. H. Burnell*	134
Hæmangioma of the Hand, Involving Phalangeal Bones, with Distinctive Radiological Appearance	*K. Hirschfeld*	136
An Anomalous Anastomosis Between the Internal Carotid and Basilar Arteries	*S. Sunderland*	140

REVIEWS—

Fractures and Other Bone and Joint Injuries	143
The Art of Surgery: A Text-Book for Students and Practitioners	143
Surgery of Modern Warfare	143
Injuries of the Skull, Brain and Spinal Cord: Neuro-Psychiatric, Surgical and Medico-Legal Aspects	144
A Complete Outline of Fractures (Including Fractures of the Skull) for Students and Practitioners	144

Editorial Notices.

ALL articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

Authors are requested to submit two typescript copies of all articles.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in indian ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

Editorial communications should be addressed to the Chairman of the Editorial Committee, 57 Collins Street, Melbourne, or to any member of the Editorial Committee. It is understood that original articles forwarded for publication are offered to THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY solely, unless the contrary be stated.

Reprints can be supplied at cost price; the minimum number is fifty copies. Orders for reprints must be given when the proof is returned.

Exchange journals should be addressed to the Honorary Librarian, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, Victoria, Australia.

Business communications and remittances should be addressed to Butterworth and Co. (Australia) Ltd., 6-8 O'Connell Street, Sydney.

The announcements appearing in this journal contribute largely towards the maintenance of the high standard of the publication. It is, therefore, requested that, wherever possible, readers will support the business houses whose advertisements appear in the journal and that, when placing their orders, they will mention THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY.

ST. THOMAS'S HOSPITAL, LONDON.—The attention of Fellows of the College is drawn to the appeal that is being made by St. Thomas's Hospital, London, for funds to rebuild the portions of the hospital that have been severely damaged as a result of air raids. Particulars of the appeal are set out on page xx of this journal, and St. Thomas's Hospital has authorized the publishers of this journal, Messrs. Butterworth & Co. (Aust.) Ltd., to receive donations on its behalf.

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

Registered at the General Post Office, Melbourne, for transmission by post as a periodical.

VOL. XI. OCTOBER, 1941. No. 2.

INDICATIONS FOR OPENING THE COMMON BILE DUCT.[1]

By T. E. VICTOR HURLEY,
Melbourne.

MUCH difference of opinion exists amongst surgeons regarding the indications upon which the common bile duct should be opened to explore its interior for possible calculi.

There is no argument if one accepts the view of Walton[(1)] that no operation for gall-stones can be considered complete unless the common bile duct is explored from within. On the other hand, experienced surgeons such as Gordon-Taylor and Burgess[(2)] have recently deplored the growing tendency to explore the common bile duct as a supplement to cholecystectomy, and assert that choledochostomy is too frequently performed.

If it were always a simple technical procedure to open the common bile duct and with no appreciable risk, there would be less difference of opinion amongst surgeons on the advisability of this procedure, as it is the most certain way of locating contained stones. Unfortunately, however, it often presents considerable difficulties, especially, for example, in stout or muscular patients, in those with enlarged congested livers and in certain types of gall-bladder disease.

Many of these patients are often very ill, and the necessary surgical procedures involved in dealing with the gall-bladder may be a sufficient tax on the patient's resources without his incurring the additional risks of being subjected to an opening of the common bile duct if this can reasonably be avoided.

Some of these risks may be briefly stated:

1. Troublesome bleeding from vessels which overlie the duct is often unavoidable, and is a serious matter, especially in jaundiced patients.

2. The region of the common bile duct is one of the most "shock-producing" areas in the abdomen, as most anæsthetists will agree. Procedures which add to shock and prolong the operation unnecessarily in sick or elderly patients, add considerably to the operative risk.

3. After the duct has been opened, it may either be drained by a tube or the incision may be sutured and internal drainage via the duodenum relied on. In either case there is some escape of bile during operation, and this continues after operation even when suture has been carefully carried out. Bile does not clot and will seep through an apparently intact suture line. This introduces an additional risk of infection and also, if the amount of bile lost from the body is appreciable in amount, it has an unfavourable effect on the general condition and nutrition of the patient.

[1] Accepted for publication on April 27, 1939.

Realizing these disadvantages, many surgeons are of opinion that in most cases they can obtain the desired information by less drastic and safer means. They therefore open the duct only to remove stones already thus detected, or in those cases in which the clinical history or the operative findings strongly suggest the presence of stones that cannot otherwise be located.

It must be admitted that calculi are often found in the duct when the clinical history has not suggested their presence. Careful inspection and palpation of the biliary tract will, however, usually give some clue to their possible presence and enable such stones to be detected without the duct being opened. It is also true that at autopsy, stones are not infrequently found in the duct, even after it has been opened and explored at operation, so that exploration cannot be relied on to detect them in all cases.

The real issue then seems to be: is the added risk of opening the duct more or less as a routine counterbalanced by those cases in which stones can be located only by this means? My experience in these cases leads me to believe that it is not, and that the following procedure is the safest and best for the majority of cases:

1. A careful analysis is made of the details of the patient's history obtained after close questioning. In most abdominal conditions, and gall-stones are no exception, this is probably our most reliable means of arriving at a diagnosis not only of stones but also of the possibility that they may be in the common bile duct.

2. Close inspection and palpation of the biliary tract and liver are carried out at operation.

In order to give some idea of the relative frequency and importance of the symptoms met with in patients with stones in the common bile duct, I have selected a series of 1,608 consecutive cases reported from the Mayo Clinic[3] in which stones in the duct were removed at operation. These represented 13·2% of all those patients from whom gall-stones were removed.

The most characteristic feature of stones in the common bile duct is the intermittent nature of the symptoms.

Intermittent attacks of colicky pain are the most characteristic symptom. Of the Mayo Clinic patients 80% had previous biliary colic requiring an opiate for its relief; 17·1% had upper abdominal pain not typically colicky, but described as dull, boring, aching *et cetera*; 2·4% had no pain at any stage.

In 73·4% (1,181 cases in all) jaundice was present. In 1,105 jaundice was intermittent with intervals of complete freedom. In 76 jaundice was persistent from the onset, but usually varied in degree. In 427 (26·5%) of the 1,608 cases the history did not reveal the presence of jaundice at any time.

In 37% (597 cases) chills and fever were present, usually associated with attacks of colic and jaundice, but in a few cases recurring chills and fever were the only symptoms present.

Nausea and vomiting occurred in 68% (1,100 cases) associated with one or more of the attacks.

The operative findings were as follows: In 1,021 (63·5%) of the 1,608 cases stones were found in both gall-bladder and common bile duct. In 587 (36·5%) stones were found only in the duct. In 149 the gall-bladder had previously been removed. In 438 the gall-bladder was present, but had no stones. In 55 stones were again removed from the duct.

The points which should be looked for in searching for evidence of stones in the common bile duct are:

1. The duct is usually enlarged, thick walled, opaque looking, yellow and adherent in varying degree to the fat of the gastro-hepatic omentum.

2. In the lower portion of its course where it is in close relation with the head of the pancreas, the latter is usually harder than normal,

sometimes to such a degree as to mask the presence of stones and to confuse the diagnosis with chronic pancreatitis apart from stones, or with carcinoma.

3. Aspiration of the duct with a hypodermic or fine exploring needle will often yield bile which is abnormal, either turbid or showing particles of débris or "mud", or pale (white bile) *et cetera*.

4. A small contracted thick walled gall-bladder with or without stones may be found.

5. The liver may be enlarged and congested.

Occasionally a plain X-ray film may show a stone in the common bile duct, and in a patient not jaundiced cholecystography may help in the diagnosis, especially if the result is positive.

Palpation of the common bile duct is usually most effectively done from the left side of the operating table with the left hand. The duct is felt between the left thumb and index finger, the latter being inserted through the foramen of Winslow. The infraduodenal part of the duct is of course the most difficult to feel because of the intervening pancreas; but with care this can be done readily and effectively in most cases.

If no stone is felt in a patient in whom either the clinical history or the appearances of the biliary tract have strongly suggested the presence of stone, then one is justified in opening the common bile duct to explore it. In cases in which the duct is greatly enlarged a finger gently inserted is the most reliable probe if the usual flexible metal probes, scoops or small rubber catheters have failed. Palpation between a finger outside the duct and a probe inside it is often effective.

Careful technique of opening and closing the incision in the duct will minimize the risks of this procedure, the chief points being the disposal of packs around the operative field, the use of a sucker, and in cases in which it is desired to close the incision and prevent bile leakage, the placing of a free omental graft over the suture line.

It is of some comfort to the surgeon to know that not all cases of biliary colic occurring after cholecystectomy are due to overlooked stones in the common bile duct. This is especially true in the first few weeks after operation when other conditions, such as the passage of biliary mud or débris or a ligature, may be the cause. Subsequent exploration of the duct in these cases is not infrequently fruitless so far as stone is concerned, and chronic pancreatitis may be found. Spasm of the sphincter of Oddi has also been invoked. It is admittedly very difficult to be sure even at subsequent operation in these disturbing cases that a stone has not been missed. In one such case a stone was subsequently passed *per rectum*, and recovered after the stools were sieved.

REFERENCES.

(1) A. J. Walton: "A Text Book of the Surgical Dyspepsias", 1923, page 527.

(2) G. Gordon-Taylor and A. H. Burgess: "Obstructions of the Common Bile Duct", *The Lancet*, Volume ii, 1937, page 446.

(3) E. Starr Judd and James M. Marshall: "Gall-Stones in the Common Bile Duct", *Archives of Surgery*, Volume xxiii, 1931, page 175.

A REVIEW OF THE RESULTS OF SUTURE OF THE COMMON BILE-DUCT AFTER EXPLORATION.[1]

By B. T. Edye,
Sydney.

During a recent trip abroad I was impressed by the fact that drainage of the common bile-duct following exploration appeared to be a universal practice and I began to wonder if the method of closure employed by Sir Alexander MacCormick and a number of his disciples, including myself, was obsolete—whether we were, in fact, out of date in our methods and were not giving those of our patients affected with stone *et cetera* in the common bile duct the best service available. I therefore thought it would be an instructive lesson to review my records and, as far as possible, analyse the results. This communication is the outcome, and I trust it will lead to a frank discussion concerning the relative methods of drainage contrasted with closure of the common bile-duct, following its exploration for whatever purpose.

On a number of occasions in the past I have discussed this question with Sir Alexander MacCormick. In his opinion, after clearance of the ducts and dilatation of Vater's papilla, it was better to adhere to Nature's method of drainage into the duodenum than to institute external drainage. He said that after practising the method for many years he had not found sufficient reason to abandon it for external drainage, except in certain extreme cases.

The technique employed by Sir Alexander MacCormick, which I will call the MacCormick operation, was described by Mr. Douglas Miller[1] in a paper read before a meeting of the Royal Australasian College of Surgeons and afterwards published in this journal.

In order that you may understand the advantages and also the disadvantages of closure of the common bile-duct, I trust you will bear with me whilst I recapitulate the main features of the operation.

After exposure of the first or supraduodenal portion of the common bile-duct a retraction suture of fine silk is inserted into its wall on each side and held with artery forceps. If possible, these sutures should not perforate the duct. Between the sutures the duct is opened by a longitudinal incision of sufficient length and is then explored. All stones *et cetera* are removed and every effort is made to render the ducts—both common and hepatic—free of foreign material. The next step is to pass a probe along the common duct to the duodenum. Following the probe, Liston's sounds, which I prefer, or Hegar's dilators are passed, with the object of dilating Vater's papilla.

When each sound impinges on the papilla a sense of resistance is experienced. This resistance is overcome by gentle pressure and the sound passes into the duodenum. No undue force is used as the duct or the duodenum may be lacerated. Disasters following this accident have been reported. The degree of dilatation varies according to the size of the duct and the freedom with which the sounds pass. When the calibre of the duct is little if at all increased the passage of a few of the smaller sizes will suffice. In others all sizes up to the largest may pass easily. There should be no doubt in the surgeon's mind that the sounds are actually passing into the duodenum. Sometimes it is difficult to be sure of this. The length of the duct, as indicated by the distance traversed by the sound, may lead one to

[1] Accepted for publication on April 15, 1939.

assume that the duodenum has been entered. But here a fallacy creeps in because the duct which has been obstructed for a considerable time may be lengthened as well as dilated. Moreover, the duct may be stretched by the pressure exerted on the sound. On a few occasions, as judged by subsequent events, I believe I have been deceived in this manner. Another source of confusion arises when a dilated, thickened and adherent cystic duct, which enters the common duct at a lower level than usual, is mistaken for the latter. The possibility need only be kept in mind to be recognized, otherwise it may keep the surgeon puzzled for a considerable time.

Surgery of the common bile-duct may be most difficult and tedious, and the patients are frequently in poor condition. But unless it is proved beyond doubt that there is a free exit for the bile into the duodenum, trouble will follow whether the common bile-duct is drained or whether it is sutured.

Should a sound fail to pass, it may indicate the presence of another stone. This is one of the most useful features of the method. Small sounds may slip past a stone, but larger ones will push the stone towards and even through Vater's papilla. If the sound still fails to pass into the duodenum, it is withdrawn and the duct is again explored for stones.

If the difficulty persists, I do not hesitate to open the duodenum while the sound is held by an assistant in such a manner as to project Vater's papilla towards its anterior wall. The duodenum is then opened by a short transverse incision directly over the projecting sound. By this means an impacted stone is removed or a constricted Vater's papilla is incised. After this manœuvre the larger sounds are passed and clearance of the common duct is assured.

It is understood that the hepatic ducts are cleared also. All surgeons are aware that stones may lie so far within the hepatic ducts as to be out of reach, and there is no method of demonstrating their presence. These stones may descend later and cause obstructive symptoms. However, if Vater's papilla has been dilated as described, there is every possibility that any remaining stones will escape spontaneously into the duodenum.

The closure of the common bile-duct after its clearance is accomplished by accurate suturing with the finest catgut on the finest needles. The more dilated and thickened the wall of the duct, the easier it is to suture. A normal duct which has been opened in a search for stones is difficult to suture neatly because of the thinness of its wall. After the closure of the duct is completed, the peritoneum which was incised to expose it is repaired with interrupted sutures. A tube, with or without a gauze wick, is now placed in relation to the duct and brought through a stab wound made at a carefully selected position towards the right. Occasionally the circumstances are such that it is better to bring the tube through a convenient part of the incision. This tube provides for the escape of any bile which may leak through the suture line in the duct. As soon as bile ceases to escape, the tube is partially withdrawn and is removed altogether on the following day. The tube is seldom retained for more than four or five days.

Sometimes it is possible to explore the common duct and to pass dilators through the cystic duct which has become sufficiently enlarged over the years by the passage of stones. Following this procedure the cystic duct is closed by ligation or by suturing. The passage of a probe through a cystic duct of small calibre along the common duct to the duodenum is apt to be misleading and should not be accepted as proof that the common duct is clear of stones.

The present investigation covers 148 patients operated upon both in public and in private hospitals, and it is concerned only with the period of time necessary for complete convalescence. In two patients the common duct

was explored twice. Of the 150 operations (see Table I) it was found in eleven instances that the cystic duct had been sufficiently dilated by the passage of stones to allow, through it, adequate exploration of the common duct and dilatation of Vater's papilla. After this procedure the stump of the cystic duct was ligated or sutured. In one patient in whom acute cholecystitis was complicated by acute pancreatitis and jaundice the common duct was explored with negative result, but not sutured, a tube being placed down to but not into it. In one extreme example of septic cholangitis, catheter drainage of the common duct was instituted and maintained for a fortnight.

TABLE I.

Sex Incidence and Mortality in 150 Explorations of Common Duct.

Patients.	Number of Patients.	Mortality.	Percentage Mortality.
Males	30	6	20·0
Females	120	8	6·8
Total	150	14	9·6

Before coming under my care twenty-four of the patients had undergone one or two operations upon the biliary system. These operations were either of the nature of cholecystostomy or cholecystectomy, and in three instances in addition the common duct had been opened and stones had been removed.

I had previously performed cholecystectomy upon five of the patients, from two of whom also stones were removed from the common duct. In one of these cases when the common duct could not be examined satisfactorily at the first operation, a stone was removed from it at the second operation. In the remaining four no stones were found in the common duct at the second operation. But in one there was stenosis of Vater's papilla, in a second a stricture of the common duct, and in two the common duct was normal.

Of the 148 patients operated upon 118 were females and 30 were males. The youngest patient was aged twenty-six years and the oldest eighty years, and there were eleven aged seventy years or over. In ten instances, in addition to opening the common duct, it was necessary to expose Vater's papilla transduodenally in order either to assist in the dislodgment of a stone or to incise the orifice of the duct which had become stenosed.

TABLE II.

Incidence of Stone in 150 Explorations of Common Duct.

Total Explored.	Stones Found.	No Stones Found.
150	88	62

Of the total number of explorations of the common duct, stones were removed in eighty-eight. In the majority of the remaining sixty-two it was obvious that stones had passed through the common duct into the duodenum as indicated by the dilatation and thickening of the wall of the duct and the ease with which the largest sounds passed through Vater's papilla. In a few one gained the impression that a stone was pushed into the duodenum by

the sounds. In a small percentage one had to assume that stones had probably never entered the common bile-duct.

Perhaps it will not be out of place here to mention the criteria which guide a surgeon in deciding when the common duct should be opened and explored.

1. The clinical history with special reference to the occurrence of jaundice, rigors and excessive loss of weight. It should be remembered, however, that a person may harbour stones in the common duct for months or years without suffering any worse symptoms than those of dyspepsia.

2. Inspection of the common duct and observation whether it is thickened or dilated.

3. Palpation of the duct. If a stone is large enough and movable, it may be distinguishable from firm nodules in the pancreas or adjacent large lymph glands.

4. A comparison of the relative size of the cystic duct and the stones in the gall-bladder.

I have not made use of the method by which bile is withdrawn for inspection from the common bile duct through a fine hypodermic needle.

In regard to jaundice there was no mention of its occurrence in the history of forty-eight patients. Stones were removed from the common duct in twenty-five of this number. No doubt jaundice may be overlooked, but it is a well recognized fact that stones may be present in the common bile-duct, and they may be even large and numerous, without causing jaundice. The bile, as it were, trickles freely between and over the stones just as the water does in a pebbly stream.

TABLE III.

Incidence of Jaundice in 150 Explorations of Common Duct.

Observation.	Number of Cases.	Stones in Common Duct.	No Stones in Common Duct.
History of jaundice	102	63	39
No history of jaundice ..	48	25	23
Total	150	88	62

In thirty-nine instances in which there was a history of jaundice, no stones were located in the common duct. In many of these there was evidence, as mentioned before, that stones had passed through. In some, no doubt, the jaundice was due to obstruction by inflammatory swelling of the common duct related to an attack of acute cholecystitis.

In the series there were twelve deaths in hospital and two deaths at varying periods after the patients left hospital. Excluding two patients in whom closure of the common duct was omitted, the death rate was approximately 9·6%.

In a considerable percentage of the patients the operative risk was increased by advanced age, impaired liver function, myocardial deficiency *et cetera*. Table IV briefly summarizes the causes of death.

From the details given in Table IV it will be noted that death could be attributed with reason to faults in the technique of the operation in three instances and doubtfully in two instances, making five in all. The remainder of the deaths were due to cholangitis, cardiac or pulmonary disease, and in one instance to cirrhosis of the liver.

TABLE IV.

Number of Case.	Sex.	Age.	Time of Death After Operation.	Remarks.	Probable Cause of Death.
I	M.	71	3rd day.	Aged and very ill; common duct difficult to clear; probably an overlooked stone became impacted in Vater's papilla; free escape of bile.	General peritonitis.
II	M.	52	3rd day.	Tube removed in twenty-four hours in error, resulting in free escape of bile into peritoneal cavity; small stone found in common duct at autopsy; this probably would have passed spontaneously.	General peritonitis.
III	M.	49	10th day.	Normal convalescence; wound healed and abdomen satisfactory. Then violent pain epigastrium and chest, collapse and death.	Coronary disease.
IV	F.	68	11th week after second operation.	First operated upon for excision of gall-bladder and removal of stones from common duct; nine weeks later again operated upon and found to have stricture of common duct; symptoms of cholangitis persisted.	Septic cholangitis and myocardial defect.
V	F.	61	7th week.	After operation, renewed attacks of pain, jaundice and pyrexia. At operation sounds did not pass well and stones probably overlooked.	Septic cholangitis.
VI	F.	59	3rd day.	Rapidly failed and died without any evidence of peritonitis. Temperature 103°. Cause doubtful; probably acute cholangitis.	Septic cholangitis.
VII	F.	62	3rd day.	Symptoms of general peritonitis developed rapidly. Cause doubtful.	General peritonitis.
VIII	F.	56	3rd day.	Two operations on biliary system previously; myocardial defect; operation very tedious; no stones found; collapsed from time of operation.	Myocardial defect.
IX	M.	60	7th day.	Severe grade of cirrhosis of liver; no stones found in common duct; small contracted gall-bladder.	Cirrhosis of liver.
X	F.	65	9th day.	Very stout, operation tedious. Wound healed and abdomen normal. Died suddenly.	Myocardial defect.
XI	F.	28	25th day.	Very ill at time of operation. Wound healed; developed septic pneumonia and pyæmia.	Pneumonia and pyæmia.
XII	M.	65	4th day.	Operation site and abdomen satisfactory. Pneumonia; calcified aortic valves *et cetera*.	Pneumonia and myocardial defect.
XIII	M.	71	5th day.	Wound and abdomen satisfactory. Pneumonia.	Pneumonia.
XIV	F.	58	1st day.	At time of operation seriously ill with septic cholangitis.	Septic cholangitis.

TABLE V.

Probable Cause of Death.

Cause.	Number.
Cholangitis	4
Peritonitis	3
Cardiac disease	3
Pulmonary complications	3
Cirrhosis of liver	1
Total	14

The greatest danger attachable to the MacCormick operation is the possibility of overlooking a stone impacted in Vater's papilla, resulting in (*a*) a free escape of bile through the suture line and a risk of peritonitis if the exit provided by the tube is insufficient, and (*b*) an exacerbation of an already established cholangitis.

I suggest that the advantages of the MacCormick operation are:

1. The thorough clearance of the common bile-duct which is possible.

2. The ability of any small stones or grit out of reach in the hepatic ducts at the time of the operation to pass through the dilated Vater's papilla.

3. The necessity of one tube for drainage and the fact that this is usually discarded within four or five days.

4. The reduction to a minimum of inflammatory thickening and adhesions in relation to the common duct as a result of the short period of drainage, so that if an operation should again become necessary its difficulties will be lessened.

5. The fact that little or no bile is lost to the patient.

6. The comparatively short stay in hospital as the ducts and the structure of the liver generally can recuperate while the patient is home and about, unhampered by the external drainage of bile.

Some of the disadvantages of the operation are:

1. The additional time needed for the suturing of the common bile-duct.

2. The danger of over-dilating Vater's papilla and lacerating the wall of the duct or duodenum.

3. The risk of the escape of bile into the peritoneal cavity immediately after the operation if the duct should subsequently become blocked by a stone overlooked at the time, and, for the same reason, the risk of accentuating an already existent cholangitis.

4. The possibility of the passage of the duodenal contents into the common bile-duct.

Of these disadvantages, 1 cannot be avoided, 2 and 3 are avoidable, and 4 to my knowledge has not been harmful.

In conclusion, I am of the opinion that suture with few exceptions is preferable to external drainage of the common bile-duct after its exploration. Such drainage is practised too frequently and is not necessary except in unusual circumstances.

REFERENCE.

(1) Douglas Miller: "Choledocholithotomy with Intraduodenal Drainage and Closure of the Common Duct: MacCormick's Technique", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, January, 1934, page 265.

HISTORICAL TRANSFUSIONS.[1]

By Eric L. Cooper,
Melbourne.

The use of human blood was early resorted to by the ancient priest physicians. The blood of a man contained the vital spirits and the soul: a weakening of the vital spirits, as in old age, or a sickness of the soul, expressed as insanity, was taken as evidence of a disorder of the blood. The blood being out of order, the philosophers advised removal of this bad blood and replacement by that of a lusty youth or an innocent lamb.

In the early days of medical practice the epileptic, the senile and the insane were given blood to drink or in some circumstances it was rubbed into their skin. Possibly the first transfusion was that given to Æson, the aged father of Jason. Medea the enchantress married Jason while he was searching for the Golden Fleece. Medea made a brew containing blood which was infused into the veins of Æson's neck, whereupon Æson became "as a young man" (*Ovid Metamorphoses*, vii, line 285). Perhaps this should not be regarded as a transfusion of blood, but rather as an intravenous infusion: there is even some doubt as to the method of administration. Æson drank some of the brew, but he was venesected from the jugular vein and some of the potion was injected into that vessel.

Argument rages over the next transfusion recorded in 1490.[1] Was Pope Innocent VIII ever transfused with blood? The patient was suffering from chronic nephritis terminating in uræmic coma. Blood is stated to have been collected from three small boys, aged ten years: each of these received a ducat (approximately ten shillings) for his blood. All three donors died before they could spend their money: death was most probably due to infection of the wound from which the blood was taken. Religious controversy at the time distorts the record of this event, and accounts differ both as to the method of the giving of the blood and its effects on the recipient. Most probably the patient drank a mixture containing blood and other stimulants. One historian states that Pope Innocent VIII died immediately afterwards in coma, others insist he lived for two years.

The possibilities and even the technique of transfusion were argued by the philosophers:[2] Andreas Libavius, a chemist, in 1615, and Robert des Gabets, a monk of Cluny, in 1653, wrote descriptions of the procedure they evolved to transfer blood from one individual to another. Probably none of these ever wielded a scalpel.

In England, William Harvey in 1628 published his discovery of the circulation. This stimulated Christopher Wren and Robert Boyle[3] in 1657 to experiment with the injection of substances into the veins of animals. When Spanish wine was instilled into the veins of a dog the animal became inebriated, howled and staggered about, and then lapsed into alcoholic coma. Opium was found to have its usual hypnotic effect. An ounce of liquid extract of opium injected into the veins of a mastiff produced a prolonged sleep lasting two days and two nights. "Afterwards the dog became fat." However, when opium was injected into cats they became mad.

The first successful transfusion of blood from one animal to another was performed by Richard Lower at Gresham College, Oxford, in 1665.[4] Lower joined the vertebral artery of one animal to the jugular vein of the recipient.

[1] Accepted for publication on October 5, 1939.

Philosophers and scientists at this period of history regarded the blood as carrying the vital humours: discussion raged as to whether the nature of the dog would be changed and if the animal would still recognize his master.

Christopher Wren had infused drugs into the veins of a criminal in London, but Jean Baptiste Denys (Dionis or Dennis), in Paris, was the first to transfuse blood into a man.[5] Denys in 1666 injected nine ounces of lamb's blood into the veins of a youth suffering from a prolonged fever. The improvement was described as "astonishing". Denys performed several successful transfusions of lamb's blood into the veins of an insane man.

In England, before the Royal Society, Richard Lower in 1667 transfused blood of a lamb into Mr. Arthur Coga, with the result that he "kept back impending insanity". Even the patient admitted he "felt better". These experiments were encouraged by Charles II of England, and the immortal diarist Samuel Pepys records Lower's transfusion at Gresham College.[6]

These earlier transfusions were used as a treatment of patients with deranged minds, but it was submitted that blood would be helpful when "a vein is severed by accident". It was even suggested that disagreement between husband and wife might be cured by a mutual interchange of blood by transfusion. In these earlier transfusions the recipient patient was always bled before or during the transfusion of blood. Even Medea venesected Æson by opening his jugular vein before her infusion. Following this theory, the deranged blood was removed, both by Denys and Lower, by bleeding their patients before they transfused the new blood. The earlier surgeons regarded the volume of the circulation as fixed, and they were attempting replacement transfusion of new lusty blood for the old.

Tragedy soon followed these first successes. Gaspar de Guyre in 1667, "transfusing a great quantity of blood into several dogs", observed that the receiving dogs "pissed blood".[5] This was the first record of a hæmolytic transfusion reaction. In Denys's fourth case, transfusion was performed several times. On one occasion "his arm became hot, the pulse rose, sweat burst out over his forehead, he complained of pain in his kidneys, and was sick at the stomach. Next day the urine was very dark, in fact black, as though mixed with the soot of chimneys."[7] The patient died at his third transfusion and Denys was charged before the Parisian courts with murder. He was acquitted of this charge, but the Parlement of Paris forbade further experiments on human beings. After further deaths the transfusion of blood was forbidden by an edict of the Pope in 1675.

Only sporadic experiments are recorded for the next hundred years: transfusion performed in ignorance of asepsis and of incompatibility of bloods could not withstand legal and ecclesiastic disfavour.

However, in England the work of Dr. Howard in Oxford (1792), Dr. Russell in Suffolk (1792), and Erasmus Darwin (1796)—the grandfather of Charles Darwin—revived interest in blood transfusion. James Blundell, obstetrician and anatomist of Guy's and Saint Thomas's Hospitals, was driven to experiment with transfusion of blood owing to a number of deaths from hæmorrhage after childbirth. The life of James Blundell (1790-1877) is a fascinating study. He was a bachelor and student of the classics. He quarrelled with his fellow practitioners and his hospital; he retired at the age of forty-eight years with a fortune of £550,000 sterling, which he had accumulated in his practice and by bequests.[8]

Blundell first satisfied himself that blood, still regarded as a living substance, was not changed during its passage through an instrument. In the year 1818 he invented a syringe with a three-way tap for direct transfusion of blood to his patients. He used this brass syringe for six transfusions. Two of these were successful, and he transfused as much as sixteen ounces of

blood to one patient who recovered. Blundell next added a water bath to delay coagulation and developed an instrument he named the impellor. The donor's blood was collected in a funnel and injected by a syringe kept warm by the water bath. Finally in 1828 he developed the gravitator, a funnel to collect the blood which in turn reached the patient by a long tube and a cannula; the whole apparatus was supported by an arm attached to a chair. Blundell performed at least ten transfusions—some of his patients were already dead before he began the transfusion—but five of his patients recovered. Blundell laid down the basic principles of blood transfusion: for success blood from the same species was necessary, blood was not changed by passage through an instrument, venous blood was as effective as arterial blood, air should be excluded from the apparatus, and the blood should not be injected too quickly.

While progress was rapid in the remainder of the nineteenth century, many fatal hæmolytic reactions and infective phlebitis in the veins of donor and recipient made the procedure too dangerous for it to become a routine procedure.

In 1859 Martin, of Berlin, reviewed fifty-seven transfusions, forty-three of which were successful. Higginson in 1857 invented the rubber syringe with ball valves which he used in seven successful transfusions. The Higginson syringe has become immortal in a lower sphere.

Animal blood was still being used by many surgeons, and fatalities were common. Cynically it was said that "three sheep were necessary for a transfusion": the lamb to give blood, the patient who received it, and the surgeon who performed the dangerous operation.

Autotransfusion had been performed by Esmarch in 1868 following Volkmann's suggestion.[9] Defibrinated blood had been used by Virchow and Bischoff in 1835, but argument raged as to the wisdom of using changed blood. Blood was still regarded as a living fluid, and whipping or otherwise removing fibrin was held to change its character; this idea was a relic of the humoral theory that blood contained the vital spirit. Thomas Smith used defibrinated blood successfully in the treatment of *melena neonatorum* at Saint Bartholomew's Hospital in 1873. Major in 1867 had used ammonium sulphite and Braxton Hicks in 1868 had experimented with sodium phosphate to delay clotting. About this period there was a veritable epidemic of blood transfusion and the results were often tragic.

During the Franco-Prussian War attempts at transfusion were numerous. War surgery naturally leads to advance in technique. The World War of 1914-1918 saw the development of the citrate technique, while the Spanish Civil War saw the use of stored blood become a routine procedure.

Landois in 1892 had suggested the use of hirudin as an anticoagulant, and Crile had developed the technique of direct anastomosis of an artery of the donor to a vein of the patient.[10] The limitations of Crile's method were obvious: a high grade of surgical ability was needed to unite artery and vein without the intervention of a cannula, and the volume of blood transferred could not be easily ascertained. It was not until the use of paraffin coated apparatus that the technique of transfusion of blood without difficulty of clotting became a simple procedure. Paraffin had been used by Bordet and Gengou in 1901, and was fully developed in the Kimpton H. Brown tube method (1913) and the syringe method of Lindemann (1913) and Unger (1915).

In 1914 Hustin in Belgium discovered the anticoagulant property of sodium citrate, and Agote in Buenos Aires in 1915 first used citrated blood in human transfusion. Lewisohn and Weil independently also developed the citrate method which, because of its simplicity, became the popular method

in the World War of 1914-1918, and has maintained its popularity. Thus the problem of coagulation during the transfusion was disposed of.

The development of aseptic surgical methods following Lister had disposed of the danger of phlebitis and other types of infection following transfusion. Transfusion reactions in the recipient were the sole remaining problems to be overcome. These reactions had resulted in abandonment of transfusion by many surgeons who had otherwise advanced the technique of transfusion. The clumping of red corpuscles in foreign serum had been recorded by Creite in 1869; Landois in 1875 described agglutination and hæmolysis and thus explained the hæmaturia of heterologous transfusion. Ehrlich and Morgenroth in 1899 showed the presence in normal human serum of hæmolysins to corpuscles of other species.

In 1900 Landsteiner and independently Shattock demonstrated the presence of isoagglutinins and established three blood groups.[11] In 1902 Decastello and Sturli added a fourth group. Jansky in 1907 outlined our present classification of blood groups, and Ottenburg in 1908 developed the clinical methods of typing blood and applied this to human transfusion. Moss in 1910 independently classified human blood and proved that isohæmolysis never occurs without previous agglutination. Thus the last danger of transfusion was removed, and it became an everyday procedure.

Throughout the history of transfusion the striking developments have been in the hands of enthusiasts. Often they have been few in number, often they have been frowned upon by the main body of the medical profession.

The science of transfusion has progressed until during the last year or so we see the development of "canned blood" technique, the establishment of "blood banks" and the use of cadaveric blood. What next? Will it be the transfusion of washed red cells to a patient requiring transport of extra oxygen or modified blood serum to patients needing proteins? Will a fluid replacing blood be developed free from the dangers of the unexplained reactions that still occur?

REFERENCES.

[1] W. M. Kerr: "A History of Blood Transfusion", *United States Naval Medical Bulletin*, Volume xvi, 1922, page 465.

[2] L. M. Zimmermann and K. M. Howell: "History of Blood Transfusion", *Annals of Medical History*, Volume iv, 1932, page 415.

[3] H. M. Brown: "The Beginnings of Intravenous Medication", *Annals of Medical History*, Volume i, 1917, page 177.

[4] M. W. Hollingsworth: "Blood Transfusion by Richard Lower in 1665", *Annals of Medical History*, Volume x, 1928, page 213.

[5] A. W. Holmes à Court: "The History of Blood Transfusion", *The Medical Journal of Australia*, Volume ii, 1927, page 528.

[6] Samuel Pepys: "Diary", November 14, 1666, November 21 and November 30, 1667.

[7] J. Bordley: "Reactions Following Transfusion of Blood with Urinary Suppression", *Archives of Internal Medicine*, Volume xlvii, 1931, page 288.

[8] H. W. Jones and G. Mackmull: "The Influence of James Blundell on the Development of Blood Transfusion", *Annals of Medical History*, Volume x, 1928, page 242.

[9] G. M. Dorrance and N. Ginsburg: "Transfusion: History, Development, Present Status and Technique of Operation", *New York Medical Journal*, Volume lxxxvii, 1908, page 941.

[10] R. L. Mason: "Preoperative and Postoperative Treatment", First Edition, 1937, page 130.

[11] F. A. Knott: "The Place of Blood Transfusion", *The Practitioner*, Volume cxxxiv, 1935, page 331.

TRAUMATIC OBSTRUCTION OF THE AXILLARY VEIN (PRIMARY THROMBOSIS OF THE AXILLARY VEIN), WITH A REPORT OF TWO CASES.[1]

By ROLLAND O'REGAN,
Wellington.

IT is generally agreed that the condition known as primary thrombosis of the axillary vein was first reported by von Shrotter in 1884. Since that time a gradually increasing number of reports and investigations of its features have appeared in medical literature. The condition has been observed and studied more frequently in continental countries. In England and America only in recent years have cases been reported, though Willan[26] reported three cases as long ago as 1918.

As far as I am aware, the only case reported in Australasia was that presented at the Auckland Clinical Society by Axford[2] in 1930. It is important that this syndrome, rare though it be, should be recognized by surgeons, as it is a definite clinical entity with a recognized course and prognosis and may be of considerable medico-legal importance. These considerations have impelled me to place on record the two following cases.

CASE REPORTS.

CASE I.—E.C., a carpenter, aged thirty-nine years, was admitted to Wellington Hospital on May 18, 1939. He gave the following history. At 2.30 p.m. on May 4, with the help of two mates, he was moving a heavy wheeled pump across a boulder-strewn beach. Attached to the pump was a wire-covered rubber hose about twenty feet long, and the three men were hauling the load by this hose running over their right shoulders. He said: "While putting extra effort into the task I felt something give in the shoulder and felt a severe pain which could be likened to that caused by hitting a finger with a hammer. After a minute the acute pain gradually subsided and left just a soreness." He carried on till 5 p.m. without inconvenience. That evening he felt increasing discomfort if he raised his arm above shoulder level. The next day the discomfort was worse and he had pain on using the arm. He worked all day, nursing his right arm and using his left arm whenever possible, and the right arm gradually swelled as the day went on. The next day the whole arm was swollen and painful, and he consulted his doctor. The arm got worse during the following four days, and a lump appeared under the armpit "which gave me the feeling that I was carrying a golf ball in the armpit".

On his admission to hospital, two weeks after the onset, the following notes were made. The patient is a well-developed, muscular, healthy-looking man. The right upper limb from the shoulder down is markedly larger than the left. The swelling does not pit on pressure. The arm is dusky in colour. The upper arm is tender to handle, with no focus of maximum tenderness. Elbow, wrist and finger movements are free. Shoulder movements are limited by pain, but the arm can be passively abducted to 90° without pain. The radial pulse is equal on both sides. There is a smooth tender mobile lump in the right axilla. There is a thickening along the course of the axillary vessels. The right axilla seems hotter than the left. The swelling extends on the anterior axillary fold. Superficial veins are not prominent. He has a good deal of subcutaneous fat.

No treatment was given other than the placing of the arm on a pillow at an angle of 45° from the side, the application of dry heat and the performance of active movements of the elbow, wrist and fingers. He was discharged from hospital on June 1—that is, after two weeks. X-ray examination of the shoulder region revealed nothing abnormal. A full blood investigation gave normal findings. The temperature and pulse chart showed nothing abnormal except a temperature of 100° F. on one evening.

[1] Accepted for publication on December 19, 1940.

Five and a half months later the patient said that after using the arm it swelled and he experienced a dull pain in the armpit and in front of the elbow and "pins and needles" in the arm. He felt the cold more in the affected arm. On examination, the right arm was still swollen. The circumference of the middle of the right upper arm was twelve and a half inches and of the left eleven inches. The circumference of the middle of the right forearm was eleven inches and of the left ten inches. A thickening could still be felt in the axilla over the axillary vessels. The superficial veins were visible over the right pectoral region and could not be seen on the other side. The right arm was still a little duskier than the left and on elevation of the arm the veins emptied more slowly. The blood pressure was equal on the two sides.

Eight months after the onset the arm was not swelling so much after use. The sensation of "pins and needles" was gone. The measurements were unaltered. The cord in the axilla was no longer palpable. The condition was otherwise unchanged.

Seventeen months after the onset the arm appeared practically normal. The chest veins were not prominent. After using the arm the patient complained of aching in the axilla, and he could not do any but the lightest work, though he had every motive to try as he was becoming destitute.

CASE II.—J.B., a farm worker, aged seventeen years, presented himself with a history that three weeks previously, the day after he had been engaged on a fencing job where he had to carry fencing posts on his shoulder some distance, he developed a pain high up on the right side of the chest and his right arm subsequently swelled. The pain was aggravated by deep breathing, coughing or jarring movements. He consulted his doctor, who noted that the veins over the right side of the chest and shoulder were prominent and the right arm was swollen. His health had not been robust for some time, and some intrathoracic pathological condition was suspected and he was referred for consultation. When seen three weeks after the onset, the whole right limb was still markedly swollen and the veins were prominent. X-ray examination of the chest revealed no abnormality. The patient played all games with the right hand, but was a partial left-hander.

Eighteen months later he had gradually increased his work during the time since his trouble began, and was then doing full work.

Five years later he reported that he was perfectly well, though "the veins on the side of my chest still stick out".

I did not have the opportunity of examining this patient personally, and I reproduce these notes by the courtesy of Mr. David Whyte and Dr. Nina Muir, who saw him at the time.

These two cases are fairly typical of so-called primary thrombosis of the axillary vein. Case II is somewhat unusual in that the initial pain was felt in the right side of the chest and was apparently of a pleural type. This pain may have been due to thrombosis in a costo-axillary vein. I have not found a case in the available literature showing this same peculiarity. Case I is more typical. It is of interest that this man received compensation under the *Workers' Compensation Act* on the ground that the condition arose as the result of an accident in the course of his employment. This directs attention to the medico-legal aspects of this condition.

SYMPTOMATOLOGY.

The Typical Syndrome.

Sufferers from this condition are predominantly males. They are usually from twenty to forty years old, and are well-developed muscular individuals in good health. The condition as a rule follows on some unusual muscular effort. In most cases the right arm is affected. When the left arm is affected the patient is usually left-handed, or if he is right-handed he has been making some unusual left-handed effort. Pain in the armpit or shoulder region is followed at a varying interval by swelling of the arm. There may be a tender cord along the course of the axillary vessels or a glandular swelling may be palpable in the axilla. Other evidence of venous obstruction, such as cyanosis and coldness of the swollen limb, is present. The radial pulse is normal. There may be tinglings and weakness in the arm and hand. Later the veins over the chest and shoulder become dilated and visible. The condition slowly

resolves and resolution may be complete in a period varying from a few weeks to many months. Prominent veins of the collateral circulation and some residual swelling of the arm may remain as permanent evidence of the venous blockage.

Variations in Syndrome.

Considerable variation in the clinical picture can occur in all details. Females are affected in a small proportion of cases. Pain may at all times be entirely absent. There may be no axillary cord or glandular swelling. There may be no history of trauma or effort whatever. Thrombosed vessels may be palpable even to the elbow. The swelling may or may not pit on pressure. There may be evidence of nerve involvement, such as weakness of grip and tinglings in the arm. The condition may clear up permanently without any disability or it may cause permanent disability. Finally, the patient may have several attacks. If there is any one explanation of the cause of this condition it should explain the clinical findings in all these variations.

NATURE OF THE CONDITION.

There is no doubt that the condition is essentially one of obstruction to the venous outflow from the upper limb. This view is generally accepted, and it is borne out by the clinical evidence. There are the swelling, cyanosis and coldness of the limb and dilatation of the collateral veins. This last-mentioned feature is vividly brought out in infra-red photographs[13] of the upper limb and shoulder region. Further, it has been shown that the oxygen content of the venous blood of the affected limb is lower than in the venous blood of the normal side.[10] The emptying time of the veins on the affected side is prolonged. Finally, the blockage in the vein has been demonstrated by venograms taken after the injection of "Thorotrast"[24] and "Abrodil".[1]

From this generally accepted view controversy begins and centres round the cause of the blockage.

ÆTIOLOGY.

All attempts to trace the ætiology of this condition to infection have failed. The evidence that has been brought forward to support an infective theory has been reviewed by Rudolph Matas,[17] who concludes: "It is evident to anyone who has given much attention to the study of this type of vascular disease that the theory of 'an infectious traumatic phlebitis' as held by [some authors] is not tenable." The Wassermann test usually produces no reaction, and careful hæmatological studies have failed to reveal any departure from the normal. The sole factor which appears consistently to play an important role is that of effort involving the use of the affected arm.

The Role of Effort.

The association of the onset of the condition with forcible use of the arm is well established. The degree of effort needed to precipitate the condition is, however, very variable and this is one of the confusing factors in an attempt to obtain a clear picture of it. The effort is commonly considerable, such as that involved in checking a restive horse, rowing in a race or the prolonged beating of clothes with a heavy stick. More frequently the effort is of a more ordinary sort, such as stirring a Christmas pudding, playing tennis or golf or doing "setting up" exercises. In other cases again the effort is trivial, as in putting jars up on a shelf, hanging curtains or shaving. Finally, there is a small number of cases in which no effort of any sort can be recalled. Such cases have been recorded by Ross,[22] Oldfield and Pyrah,[20] Taylor,[23] Veal and McFetridge[24] and others.

In the cases here recorded the efforts were such as are common among working men, though they appear severe efforts for others and would be more than severe for women or sedentary workers. In general, the efforts which have been reported as preceding the onset of this condition are really quite ordinary efforts for the class of person concerned. Further, in a not inconsiderable number of cases the effort is quite trivial. This point is of some importance because, if the condition is due to injury to the axillary vein as a result of effort with the arm, the most striking thing is that such efforts are extremely common and this injury is very rare.

To summarize: (*a*) Effort is the only established ætiological factor. (*b*) Efforts which produce this rare lesion are extremely common. (*c*) The injury occurs typically in young healthy athletic persons.

One is compelled by these three facts to conclude that if these individuals damage their axillary veins in these ways they must have axillary veins that differ from those of the remainder of mankind. That is, that the veins are anatomically different from the average in such a way as to make it possible for these efforts to produce damage. This conclusion is made all the more likely when it is remembered that the axilla is an area of great anatomical variation, and that these variations affect not only the axillary vessels and their tributaries but also the brachial plexus, lymph nodes, muscles and fasciæ. Further, the size and shape of the axillary inlet and the proportions of the axillary cone vary in different physical types. That the real reason why a common effort should damage the axillary veins of some persons would be found in an anatomical peculiarity which made it possible, was the conclusion of Veal and McFetridge[24] after their very interesting study of the mechanical effects on the axillary vein of movements of the arm. They conclude that the precise nature of the defect will remain unknown until the opportunity arises for autopsy examination in a case of this kind. It might be pointed out in passing that if this is true, studies on the anatomy of the average axillary vein and the influence of mechanical factors upon it can be of limited value only in explaining the cause of injury in these cases.

If this view is correct it offers an explanation of the varying degree of effort concerned in the reported cases. Some persons are more anatomically prone to injury than are others. In one a great effort is needed and in another very little. It may be asked, however, why in a person, say, twenty years old a simple arm effort causes the injury when the patient must have made similar efforts hundreds of times before in his life. For example, in Puhl's[21] case of the man who met with this accident when emptying corn sacks, why did the injury occur as he emptied the fifteenth sack and not the first one? The answer must be that only with this particular effort were the mechanical factors concerned those required to produce the injury. Factors to be considered are the distension of the vein, the rate of blood flow, the venous pressure, the respiratory phase, the direction and force of the movement and the perfection of the coordination of the movement. All these factors are variables. Prolonged use of the arm increases the venous return by the vein, causing distension and a raised pressure in the vein. It has been shown that on each inspiration there is a check to the outflow from the vein. On deeper breathing there is a back-flow to the vein, and on straining this back-flow may even reach the veins of the arm. Further, the rate of emptying of the vein varies with the position of the limb.[24]

To sustain this injury, then, it is probably not sufficient that a person should be anatomically prone; in addition a suitable combination of these variable factors is also necessary. In attempts to elucidate the problem, several very interesting studies have been made on mechanical factors which operate on the axillary vein in health. These may be summarized as follows.

Anatomical Studies.

Lowenstein[16] made dissections of the axillæ of thirty cadavers and studied sections of six others. He concluded that pressure on the vein by the costo-coracoid ligament and subclavius muscle with the arm in abduction during forced expiration caused the damage to the vein.

Gould and Patey[8] injected plaster of Paris into the axillary veins of cadavers and in two instances observed a broad deep groove caused by the subclavius muscle, and in another a shallow groove at the site of the costo-coracoid ligament. At this level they demonstrated a bicuspid valve in the vein as a constant anatomical feature. They regarded a rupture of this valve as the fundamental lesion causing thrombosis.

Veal and McFetridge[24] studied the vein by means of radiograms taken as "Thorotrast" was injected. They showed that in hyperabduction and external rotation the vein is stretched at the point where it crosses the subscapularis muscle, and this observation was confirmed by a study of fresh autopsy material. They also made other important observations referred to above.

These researches give strong hints as to the probable mechanism of the vein injury, but they cannot afford a final explanation. All explanations based upon them do not explain why the lesion is so rare. This fact of the commonness of the alleged cause and the rarity of the lesion compels the conclusion stated above, that a person must be anatomically prone to the injury, and a study of the anatomically non-prone majority cannot be expected to give a final explanation of the mechanism of the injury.

Nature of the Obstruction.

The question still remains as to how the injury to the vein by over-distension, stretching of the wall or tearing of a tributary causes the vein to be blocked. Theoretically the cause may be external to the vein, in the lumen or in the wall.

1. *External Pressure on the Vein—for example, Hæmatoma.*—It is probable that hæmatoma from a torn tributary vein is sometimes at least a contributory factor in causing the signs of blockage of the main vein. Matas,[17] whose access to foreign literature has been free, states: "A perivascular hæmatoma is sometimes found (at operation) which is caused by rupture of the small tributary vessels of the axillary." He also quotes a case reported by Moure and Martin (1932)[18] in which a patient who showed the typical syndrome was operated on two months after the occurrence because there was no improvement. They found the vein wall normal and no thrombosis was present. "The inner border of the coracobrachialis was found ecchymotic, suggesting the existence of a hæmatoma which had been absorbed, leaving a considerable serous collection in the intramuscular spaces. Nothing was done. The wound was cleansed of serum (which was sterile) and closed without drainage." The patient was completely recovered and at work in three weeks.

It is probable that the swelling which is sometimes found in axilla and resembles an enlarged gland, is sometimes in reality a hæmatoma. In only one recorded case have I found reference to discoloration round the anterior fold.[6]

The tinglings and other paræsthesiæ so commonly complained of may in part at least be due to irritation of fibres of the brachial plexus by such a hæmatoma. Further, the fact that the swelling in the arm sometimes does, and sometimes does not, pit on pressure may be due to varying degree of lymphatic as well as venous obstruction from the same cause.

2. *Obstruction in the Lumen—that is, Thrombosis.*—There is no doubt that actual thrombosis is not present in all cases. This has been proved conclusively by many observations made at operation. Thrombosis is therefore not an essential factor in the causation of the blockage. It may not occur, and apparently it modifies the clinical picture not at all in that cases are reported with and without thrombosis which are clinically identical. It cannot be assumed that if a thickening is palpable along the course of the axillary vein thrombosis has occurred in the vein. Only where the thrombosis extends into the subcutaneous basilic vein can its presence be surely diagnosed. This has been reported frequently, but among the reported cases available I have not found one in which the thrombosed basilic vein was observed soon after the onset. Thus in Gould and Patey's[8] series of eight cases thrombosis of the basilic vein is recorded in three cases, but they were first seen three weeks, twelve days and two weeks respectively after the onset of symptoms. Among Barker's[3] six cases it was reported in one and this one was first seen one week after the onset of symptoms. Another fact worth considering in this connexion is the absence of embolism. If thrombosis were the usual cause of the blockage it would not be surprising if embolism were to occur at least occasionally when the arm was not rested. However, embolism does not occur, and this suggests that thrombosis occurs distal to the blockage and is not an essential feature of the condition. Thrombosis must properly be regarded as a result of the blockage and not its cause. For this reason the condition is wrongly called "primary thrombosis of the axillary vein", and I suggest that in the present state of knowledge the name used in the title of this paper is more appropriate.

3. *Obstruction in the Vein Wall—for example, Extravasation into the Vein Wall or Venospasm.*—Obstruction in the vein wall is probably the commonest cause of blockage. It is probable that extravasation occurs into the wall of the vein or between the vein and its sheath, and that this causes the vein to be narrowed and the narrowing is made permanent by late fibrosis. In cases in which the intima is also damaged, thrombosis may complete the block. In a recent important paper Puhl[21] describes his finding in two cases treated by operation. In one the vein was exposed after symptoms had been present two and a half months and improvement had ceased. The second patient was operated on for the same reason eight months after the injury. In neither case was thrombosis present. He found the vein to be narrowed and the vein wall thick like the wall of an artery. The distribution of the narrowing was patchy, and between thickened segments the vein wall was of normal thickness. By contrast with the narrowed parts the normal portions had the appearance of varices. In one case the perivenous fascia was densely adherent to the vein wall, and in the other it could be freely dissected away. Perivenous tissue removed was by culture methods found to be free from pathogenic organisms. From a microscopic study of this tissue he concluded that there was present an aseptic fibrosis following injury. Both patients benefited when the vein was freed from the fascial investment.[1]

Much interest has been shown in all recent articles in the view widely held among continental writers that blockage is caused by severe local spasm of the vein wall. It is claimed that a result of injury, irritation of a perivenous sympathetic plexus, occurs and causes the local spasm of the

[1] Illustrations taken from this paper showing the condition found at operation were reproduced in a recent number of this journal.[12] The review which includes these pictures is in many important particulars inaccurate.

vein wall. The names of Cottalorda[5] in France and Löhr[15] in Germany are especially associated with this view. Matas[17] presents Cottalorda's view as follows:

> The syndrome which we recognise clinically as thrombo-phlebitis by strain is dependent on vascular lesions in which the vein predominates. These lesions have their origin in a venospasm induced by sympathetic irritation caused by trauma. . . . The mere obstruction of the lumen is not the only cause of the œdema, but it is the persistent irritation of an inflamed or injured perivenous plexus that is responsible for the rapid œdematous swelling.

Holding this conception of the essential character of the condition, these writers advocate a perivenous sympathectomy or actual excision of the occluded segment as a logical treatment. Reports claiming brilliant results from these procedures in long-standing cases have been published. Unless one is willing to accept the theory outlined, it is not easy to offer an explanation of such results. Leriche's views on the sympathetic innervation of peripheral arterioles, of which this theory appears to be an adaptation, have been seriously challenged on anatomical grounds.

It would be of interest to study at first hand the reports of these writers. Unfortunately access to continental papers is not at the moment easy to obtain. The conception of a local spasm of the vein wall prolonged over a considerable period, and in some cases recurring, is not a familiar one. Veins receive some harsh treatment nowadays. Irritating substances of all sorts, from "Neoarsphenamine" to calcium salts and sclerosing agents, are injected into them and sometimes around them and no local spasm is observed. Veins are handled and ligated at operation and no spasm is observed. The axillary vein is stripped of its branches and its fascial coverings are peeled away in the radical operation for carcinoma of the breast, and no spasm is observed. Further, the condition is not paralleled by the behaviour of any other injured vein. It is curious that the lesion should occur solely in this deeply placed vein and as a result of this rare injury.

TREATMENT.

There is no doubt that with rest to the arm in abduction and simple physical therapy and elastic bandaging most patients will recover, and this line of treatment should be adopted as long as improvement is maintained. When operative treatment is undertaken before the condition has become stationary, it is impossible to assess its value or even to say whether it is of any value at all. When a patient's condition fails to improve up to a point which gives him a useful arm, the question of operative treatment arises. The danger of operating would appear to lie in the possibility of damage to the collateral circulation, and operation should not be undertaken unless the disability is of a degree to warrant it. Other considerations, however, should encourage intervention. The vein may prove to be subjected to external pressure, as in the case of Moure and Martin[18] quoted above, in which mere exposure in the vein was followed by complete recovery in three weeks. If at operation the vein is found to be stenosed by fibrosis, but not completely occluded, it would be rational surgery to strip, if possible, the constricting sheath. This is in effect what was done in the cases of Puhl quoted above, and though recovery was not perfect, considerable improvement followed, whereas previously the condition was stationary. If the vein is found to be completely occluded, the occluding segment should be resected first on the ground that this procedure cannot increase the blockage which is complete, and secondly because this procedure has in some hands given brilliant results. In the present state of knowledge, however, it is not justifiable to resect any vein which is not completely occluded, as there must

be a risk of making the condition worse. This is the view that Puhl took when he stripped the perivenous fascia in his two cases, not regarding resection as justified, and I think that it is the proper view.

PROGNOSIS.

Prognosis as to life is uniformly good. In regard to the function of the arm the majority of patients recover sufficiently to do full manual work, but there are exceptions. The degree of recovery must depend on whether the vein is totally occluded, on whether it is permanently occluded, on the efficiency of the collateral circulation, and on the degree to which tributary vessels are involved. Permanent partial incapacity is exceptional, but it does occur as in the cases reported by Matas,[17] Horton,[10] Barker[3] and others. Where, as is usual, functional recovery is complete, the period of disability is very variable. This period varies from many months to as little as one month, as in the case reported by McIntosh.[19] For years afterwards there may be minor troubles, such as swelling of the arm after it has been used and discomfort in the axilla after the arm has been used elevated above shoulder level, and permanent prominence of the veins over the chest with some thickening of the limb is not unusual. This does not, however, interfere with full use of the limb.

Cases are also reported in which the condition has recurred.[3][13][17] This is exceptional, and is probably due to further injury to a vein which has been incompletely occluded by the initial injury. To this the patient may have been rendered more liable by increased fixation of the vein as a result of perivenous fibrosis. When the vein has been finally occluded by an organic cause a recurrence of acute symptoms should be impossible, and with an adequate collateral circulation the patient should suffer no further disability. If this view is correct relapse will not continue indefinitely, but only until the vein is permanently occluded. It is reasonable to conclude, therefore, that patients showing indubitable evidence of thrombosis, such as the extension of the thrombus into the basilic vein, are not likely to have recurrent symptoms.

Case I here reported seems to me to be one in which relapse is very liable to occur. The patient has only minor obstructive symptoms now. Most of the blood must leave his arm by the axillary vein. He has a prolonged aching over the vein after use, which prevents him from working. I visualize the condition as a patent vein of reduced calibre fixed by perivenous fibrosis. These are probably the conditions conducive to relapse.

Which patients are going to recover completely and which are not cannot be forecast accurately. The degree of trauma causing the damage is no guide. The severity of the condition and the rate of recovery over the first three months give the only clue. If at the end of this time progress is stationary or discouragingly slow, it should be remembered that it is claimed that operation has given complete and rapid cure in some cases.

SUMMARY.

1. Two cases of traumatic obstruction of the axillary vein are described.
2. The nature and ætiology of the condition are discussed.
3. The available literature on the subject is reviewed.

ACKNOWLEDGEMENT.

It is a pleasure to acknowledge the assistance given in the preparation of this paper by the librarian of the Gordon Craig Library, who has made available reprints and photostatic copies of papers otherwise unattainable.

REFERENCES.

(1) O. Andersson: "Venography in a Case of So-Called Traumatic Thrombosis of the Axillary Vein", *Acta Radiologica*, Volume xix, 1938, page 126.

(2) M. Axford: "*The New Zealand Medical Journal*, Volume xxix, 1930, page 379.

(3) N. W. Barker: "Axillary Thrombophlebitis Caused by Strain or Effort", *Proceedings of the Staff Meetings of the Mayo Clinic*, Volume x, March 6, 1935, page 156.

(4) H. M. Clute: "Idiopathic Thrombosis of the Axillary Vein", *Surgical Clinics of North America*, Volume xi, April, 1931, page 253.

(5) Cottalorda: Quoted by Matas, *loco citato*.

(6) J. Fettes: "Thrombosis of Axillary Vein", *The British Medical Journal*, Volume ii, 1933, page 758.

(7) D. Firth and R. Mackay: "Primary Thrombosis of the Axillary Vein with Recurrence", *The Lancet*, Volume ii, 1932, page 679.

(8) E. P. Gould and D. H. Patey: "Primary Thrombosis of the Axillary Vein: A Study of Eight Cases", *The British Journal of Surgery*, Volume xvi, 1928-1929, page 208.

(9) G. E. Gask and J. P. Ross: "Surgery of the Sympathetic Nervous System", 1934, page 42 *et sequentes*.

(10) B. T. Horton: "Primary Thrombosis of the Axillary Vein", *The Journal of the American Medical Association*, Volume xcvi, 1931, page 2194.

(11) F. W. James: "Primary Thrombosis of Axillary Vein", *The British Medical Journal*, Volume ii, 1933, page 588.

(12) "So-Called Primary Thrombosis of the Axillary Vein (Intermittent Claudication of the Axillary Vein)" (abstract of four papers), THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume ix, 1939-1940, page 204.

(13) T. Kaplan: "Thrombosis of the Axillary Vein: Report of Five Cases with Comments on Etiology, Pathology and Diagnosis", *The Journal of the American Medical Association*, Volume cx, 1938, page 2059.

(14) T. Kaplan and A. Katz: "Thrombosis of the Axillary Vein: Case Report with Comments on Etiology, Pathology and Diagnosis", *The American Journal of Surgery*, Volume xxxvii, 1937, page 326.

(15) W. Löhr: Quoted by Puhl, *loco citato*.

(16) P. S. Lowenstein: "Thrombosis of the Axillary Vein: An Anatomic Study", *The Journal of the American Medical Association*, Volume lxxxii, 1924, page 854.

(17) R. Matas: "On the So-Called Primary Thrombosis of the Axillary Vein Caused by Strain: Report of a Case with Comments on Diagnosis, Pathogeny and Treatment of this Lesion in its Medico-Legal Relations", *The American Journal of Surgery*, Volume xxiv, 1934, page 642.

(18) Moure and Martin: Quoted by Matas, *loco citato*.

(19) J. N. McIntosh: "Primary Thrombosis of Subclavian Vein", *The British Medical Journal*, Volume ii, 1933, page 1143.

(20) M. C. Oldfield and L. N. Pyrah: "Thrombosis of the Axillary Vein", *The British Medical Journal*, Volume ii, 1936, page 68.

(21) H. Puhl: "*Zur Frage der sogennanten Thrombose der Vena axillaris*", *Archiv für klinische Chirurgie*, Volume cxc, 1937, page 569.

(22) J. C. Ross: "Primary Thrombosis of the Axillary Vein, with Report of a Case", *The British Medical Journal*, Volume ii, 1933, page 525.

(23) C. H. S. Taylor: "Primary Thrombosis of the Subclavian Vein", *The British Medical Journal*, Volume ii, 1933, page 818.

(24) J. R. Veal and E. M. McFetridge: "Primary Thrombosis of the Axillary Vein: An Anatomic and Roentgenologic Study of Certain Etiologic Factors and a Consideration of Venography as a Diagnostic Measure", *Archives of Surgery*, Volume xxxi, 1935, page 271.

(25) W. Wagner: "*Beobachtungen und Behandlung bei der sogenannten Achselvenenthrombose*", *Zentralblatt für Chirurgie*, Volume lxv, September 24, 1938, page 2169.

(26) R. J. Willan: "Three Cases of Axillary Vein Obstruction", *Edinburgh Medical Journal*, Volume xx, 1918, page 105.

A MALFORMATION OF THE HIND END OF THE BODY.[1]

By SYDNEY SUNDERLAND,
Department of Anatomy, University of Melbourne.

MALFORMATIONS of the hind end of the body are by no means uncommon, but the findings in the case about to be described are so remarkable (in fact, none of a similar nature could be found in the records) and the information they provide concerning the development of the hind end of the body is so significant, that it is felt they warrant recording. For permission to examine the specimen I am indebted to the staff of the Children's Hospital, Melbourne.

CASE HISTORY AND POST-MORTEM FINDINGS.

The patient was admitted to hospital three hours after birth, with a diagnosis of imperforate anus, and survived for nine hours. Clinical examination revealed a cyanosed full-term female baby weighing eight pounds, and did not reveal the presence of any other abnormalities apart from the absence of an anus. There was no sign of any anal dimple. The *labia minora* and clitoris were hypertrophied and projected from between the *labia majora*. No urethral orifice could be detected, whilst the vaginal orifice, which was a large for a child of its age, showed a perforate hymen through which a watery meconium exuded. The abdomen was distended, though no mass was palpable. A catheter was passed up through the vaginal orifice and the tip could be palpated inside the abdomen at the level of the umbilicus. A small amount of meconium came away, indicating a natural passage into the bowel.

When the abdomen was opened at post-mortem examination a grossly dilated and distended portion of the bowel was seen to fill almost the entire abdominal cavity. This part was thin-walled, semilunar in shape, with its long axis lying transversely across the abdomen; the convexity was directed upwards and the concavity downwards. Passing downwards and forwards from the mid-point of the concavity to become continuous with the bladder neck was a short tube which had been twisted through a quarter turn in a clockwise direction. The abdominal portion of the œsophagus, the stomach and the duodenum were normal in position and size. What appeared to be small intestine occupied the upper and central part of the abdominal cavity posterior to the dilated bowel. From this region it passed over into the left hypochondrium, from which position it passed down the right side to the right iliac fossa, where it turned to enter the right horn of the dilated bowel already described. Just below this communication the latter gave off a small pouch-like diverticulum resembling the caecum in shape and position. The true pelvis was occupied by the bladder neck and fibro-fatty tissue, whilst all the pelvic viscera were up in the false pelvis and abdomen. The kidneys were normal and in their customary position. The ureters passed down to terminate in the bladder neck. What appeared to be the Fallopian tubes were thin and coiled laterally, but abruptly became thicker and straight and, after a short course, terminated medially in a mass of tissue about the bladder neck as well as in a small centrally placed organ, resembling the uterus in shape, which was attached firmly and completely to the postero-inferior surface of the bladder immediately beneath the opening of the bowel into that viscus. The vagina was apparently absent and the tubes passed evenly over into the uterus-like body. No other intraabdominal abnormalities could be detected. The post-mortem examination had unfortunately to be performed hastily and incompletely and only the following parts could be obtained for study. The alimentary canal was removed from about the junction of the jejunum and ileum onwards and a clean sweep made of the pelvic structures, kidneys and ureters. The region about the *symphysis pubis* was excised with the pelvic structures and with it the *labia minora*. (See Figures I and II.)

Detailed Description of the Malformation.

The Alimentary Canal.

The external features of the malformation are illustrated in Figures I and II.

The tubular bowel (A) was constricted above, but gradually increased in diameter as it passed downwards to narrow again before entering the right horn of the dilated

[1] Accepted for publication on February 21, 1941.

bowel (D), which gave off a pouch-like diverticulum (C) just below the site of entry of the tubular bowel. The dilated semilunar portion measured 13·5 centimetres transversely and 8·0 centimetres from the mid-point of the concavity to the corresponding point of the convexity. The muscle coat was in a complete sheet in all parts of the

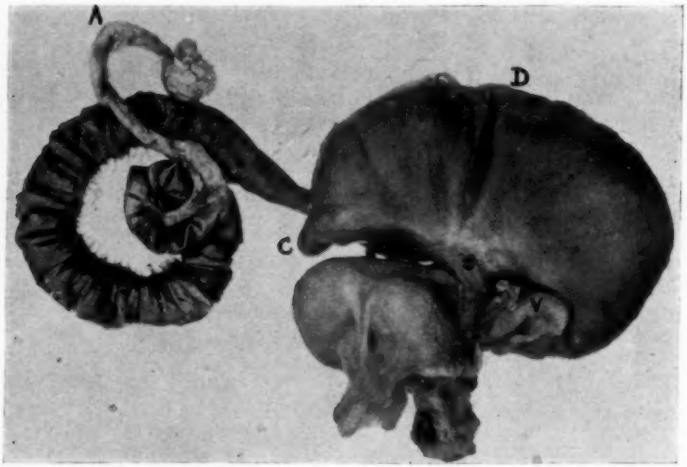


FIGURE I. Anterior aspect of the specimen as removed at post-mortem examination. A = small intestine; C = caecum; D = large intestine; S = communication between colon and bladder; V = uterine tube.

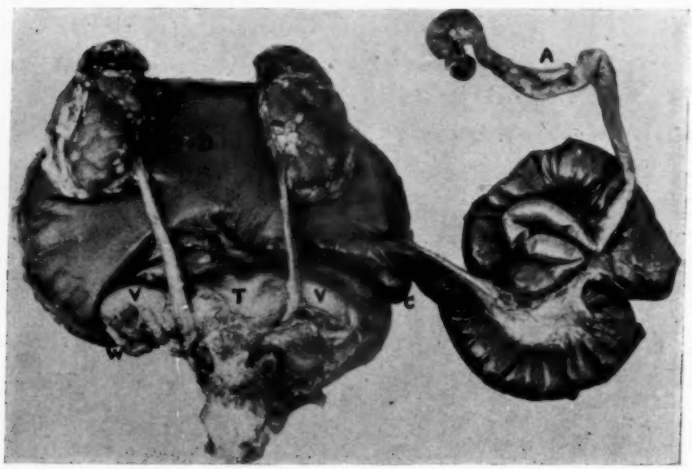


FIGURE II. Posterior aspect of the same specimen. A = small intestine; C = caecum; D = large intestine; S = communication between colon and bladder; V = uterine tube; T = vagina; W = Fallopian tube.

bowel, there being no condensation of the outer longitudinal coat into bands in any region. Two deep strands of tissue were directed across the dilated bowel from the region of the communication with the bladder. In the upper part of the bowel the blood vessels in the mesentery were arranged in arcades, but this gave way to a longitudinal arrangement in the most distal part of the tubular bowel. In the dilated portion the chief vessel ran close to, and parallel with, the convexity. The contents were a thick

viscous meconium. The mucous membrane of the tubular bowel was thrown into irregular circular folds above, but below and in the dilated portion folds were absent. The opening of the tubular into the dilated semilunar portion of the gut strongly resembled a normal ileo-caecal valve, having upper and lower lips which met and tailed off anteriorly and posteriorly to enclose the lumen of the diverticulum, thereby forming a circular lip which actually separated the diverticulum from the distal portion of the bowel. The lumen situated at the mid-point of the concavity was oval in shape and measured about 20 millimetres in its greatest diameter. A probe showed it to connect by means of a short neck (S) with the bladder. There was no definite line of division macroscopically between the small bowel and colon, though the findings suggested that the dilated portion was colon, the diverticulum caecum, and the tubular bowel small intestine. This was confirmed by histological examination. There was no trace of an appendix.

Appearing on the posterior surface of the neck-like communication, linking gut and bladder, were two well-marked folds which diverged, as if from an apex in the bladder, as they passed backwards into the bowel. The left fold, which was the better

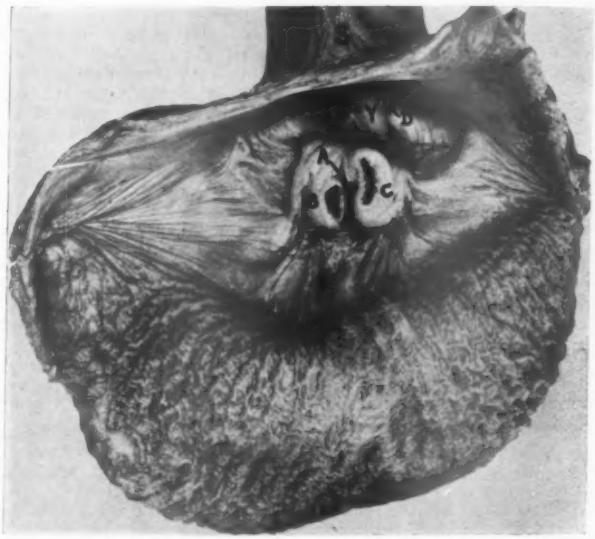


FIGURE III. Internal aspect of the base of the bladder. X and Y = fibrous folds passing from the bladder to colon; D = orifice leading to the colon; S = communicating neck between colon and bladder; B and C = vaginal orifices; A = cloacal outlet.

developed of the two, passed up on the left side of the neck and so to the anterior surface of the colon, which it then crossed close to the concave margin to terminate on the anterior surface of the caecal diverticulum. This fold was drawn out to such an extent that a deep fossa was present under its left side. The right fold passed directly to the posterior surface of the colon and was lost on that surface about half-way between the lumen and the caecum. The fibres in these strands were distinctly submucous. This was confirmed histologically when they were also found to be composed of fibrous tissue only. The colon between these strands and the opening of the neck into the bladder was lined only by tall columnar epithelium, whilst elsewhere its mucous membrane showed the typical large bowel arrangement.

The Genito-Urinary System.

The kidneys and suprarenals were normal. Double ureters were present on each side and these passed forwards beneath the tubes to open separately into the bladder. The dome of the spherically shaped bladder was faintly bilobed and a urachus arose from the medial end of the crest of each lobe. The bladder was filled with a mixture of gelatinous mucus and meconium. A number of openings were seen on its floor, and

the mucous membrane was thrown into irregular folds (see Figure III). No definite trigonal area was outlined, although the mucous membrane about the openings at the base was less wrinkled than elsewhere. Histological examination of the wall revealed normal bladder tissue. Centrally situated was an opening (A) which led out into a single tube, approximately 3·5 centimetres in length, which passed downwards and forwards beneath the *symphysis pubis* to open between the *labia minora*. The nature of the communications to be described left no doubt that this channel functioned as a cloacal canal. On section it was found to be lined by stratified squamous epithelium. Posterior to and above this cloacal orifice was an oval opening (D) which led into the twisted tube (S) (see Figures I and II) connecting with the dilated colon. This communication was a free one, no valvular arrangement being present to influence the passage of material in either direction, although it is possible that prolapse, and the twist already described, may have inhibited the free passage of contents from the distal portion of the bowel into the bladder and so have contributed to its dilatation. The two folds (X and Y) previously described in connexion with the dilated colon, were now seen to focus on the region of the orifices at the base of the bladder. On either side of the cloacal outlet (A) were two well-defined orifices (B and C) with raised thickened margins, whilst antero-lateral to these were the openings of the ureters, the upper ureter entering medial to the lower.

Attached to, and forming part of, the postero-inferior wall of the bladder, below the opening of the colon into that viscus, was the pyriform shaped body (T) (Figure II) which so closely resembled a uterus; there was no suggestion of a division of this organ into body and cervix. Arising from the supero-lateral angles of this body, and closely bound to the bladder wall at that site, were the blunt thickened tubes (V); these, after a short course of about four centimetres, suddenly decreased in size to form a very narrow coiled tube (W), suspended from beneath which was the ovary. The arrangement suggested that the pyriform organ was the uterus and the short thick tubes remains of the unfused Müllerian ducts, whilst the smaller lateral coiled portions were the Fallopian tubes. Just how erroneous these temporary conclusions were will be seen. No remains of the Wolffian ducts could be found.

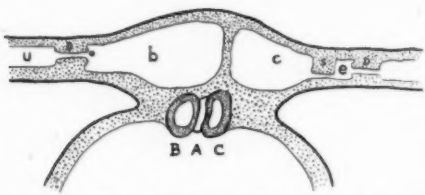


FIGURE IV. Diagrammatic representation of the condition found on dissection of the bladder-neck region. *b*, *c* and *e* = vaginal cavities; U = cavity of the uterine tube; D = cervix; O = external os; S = block of tissue projecting into the left vaginal cavity and dividing it into its two compartments *c* and *e*.

The arrangement observed when the structures about the base of the bladder were dissected is diagrammatically represented in Figure IV, which represents a horizontal section across the bladder neck just above the level of the bladder orifices (A, B, C). When the pyriform shaped organ (T) was opened along its posterior wall, its mucous membrane was seen to be thrown into many longitudinal folds. The cavity so exposed, marked *b* in Figure IV, was blind below, whilst its supero-lateral angles were expanded into recesses. Centrally situated in the anterior wall was a slit-like opening which connected with the orifice (B) to the right of the cloacal outlet of the bladder; while a second orifice (O), which exactly resembled an external *os uteri*, led from the right superior recess into the right tube (V). The left superior recess was not as marked as the right and was blind.

A probe passed through the left-sided vesical orifice (C) led into a smaller chamber (*c*) in the left tube (V), and this connected laterally beneath a ledge of solid tissue (S) with another small compartment (*e*), which in its lateral recess showed a condition identical with that found in the right recess of the cavity (*b*) of the pyriform-shaped organ (T). The mucous membrane of both these compartments was thrown into irregular folds.

Histological examination revealed the following interesting condition. The cavity (*b*) of the pyriform organ (T) and the two compartments (*c* and *e*), associated with the left tube (V), were vaginal in structure, the vaginal mucosa passing onto the raised lips of the two vesical orifices, to give way abruptly to bladder mucosa at the outer rim of the lip. The thick tubes (V) showed a well-marked subdivision into two parts. A central portion (D), which was long and lined by a well differentiated cervical mucosa showing many mucous glands, terminated medially by projecting into the vagina as the external os (O), and laterally into the remaining part of the tube as an internal os. The lateral part of each tube (U) was lined by definite uterine mucosa, whilst the finer coiled tubes (W) proved to be Fallopian tubes.

Summarizing these findings we have a condition in which the alimentary canal opened into the urinary sac. The genital system showed two uterine tubes and two vaginæ. The latter were fused over part of their course, but remained separate, the septum between the two not having broken down. The two vaginæ opened into the bladder separately, though adjacently, below the orifice of the colon. Antero-lateral to the vaginal orifices were those of the ureters whose developmental site was therefore above that of the genital ducts. Thus the gut and genito-urinary ducts all opened into a common cloacal chamber, described in this account as the bladder, and from this a cloacal canal passed to open at the perineum. The arrangement of the bowel represents a condition in which growth of the distal limb of the primary mid-gut loop has been inhibited and where development has been interrupted subsequent to the reduction of the mid-gut. Hence the external appearance of the specimen was no guide to the ultimate condition found on dissection and histological examination.

DISCUSSION.

One of the most pertinent questions arising for discussion is whether any intrauterine upset will merely arrest or delay a certain region at a particular stage of its normal development, or whether it will promote abnormal development in the part. It is well known that the former does occur, as, for example, in failure of rotation of the gut or in non-descent of the testes. Moreover, in this matter of arrested normal development, can it justifiably be said that the development is held up at some phylogenetic milestone of the animal under consideration? It may be stated definitely at this stage, and without further elaboration, that the principle of ontogeny recapitulating phylogeny can be invoked provided only basal generalized forms and not specialized offshoots of our phylogenetic ancestors are considered. Once relegated to this position, the principle loses much of the significance which many investigators have assigned to it in the past. That abnormal development can, and does, take place is equally certain, as is amply confirmed by the study of the material in teratological collections. In considering any abnormality, then, one should, if possible, attempt to ascertain which process has been at work; otherwise the use of embryological abnormalities as a means of eliciting normal stages of development is likely to be productive of much error. If this particular abnormality had its evolution along abnormal lines, then it no longer requires consideration as possibly elucidating the normal development of the hind end of the body. If, however, it represents a stage of arrested normal development, and that is the attitude which will be adopted here, then it will, perhaps, yield some information on the controversial question of the development of the hind end of the body.

Before an explanation of this unusual case is submitted, an outline will be given of the main conclusions concerning the separation of the cloaca into its urogenital and rectal compartments during embryonic development, about which there is no agreement of opinion.

Pohlman (1911), Tourneux (1893) and Felix (1912) all account for the division of the cloaca by the downgrowth of a coronal septum. According to Felix, whose account may be taken as representative of the opinions expressed by this group, the separation of the cloaca into a dorsal or rectal compartment and a ventral or urogenital compartment "takes place by the saddle between the cloacal opening of the intestine and that of the allantois growing as a partition downwards from above into the lumen of the cloaca, parallel to its dorsal wall". This partition he calls the *septum urorectale,* and referring to its formation he continues: "The middle line of the septum, however, never shows a raphe, such as would be formed by the union of the folds, and therefore the description of the *septum urorectale* as unpaired is more correct."

The principal defect in this theory is that the authors do not attempt to explain what causes the septum to develop or migrate in the fashion ascribed to it. On page 791 of Keibel and Mall's "Manual of Human Embryology" Felix states that "the lower sagittal portions of the two urogenital folds unite throughout their whole length in the median line; the extent of the union may be lessened or quite prevented by an abnormal influence on the part of the intestine. This gives a possibility for the explanation of double formations of the uterus." An examination of this statement suggests that Felix is of the opinion that the formation of the septum is not due to the union of the two urogenital folds across the mid-line, since, if fusion of the folds from below were responsible for the formation of the septum, the latter would necessarily have to develop as a bilateral partition which, it is specifically stated, it does not do. Again, the genital cord is not formed by the union of the two urogenital folds until the twenty millimetre stage is reached, and by that time the urorectal septum is said to have completely divided the cloaca into its two parts. Evidently formation of the septum and fusion of the folds are to be regarded as two separate phenomena. There is, however, no indication as to whether fusion of the folds takes place unrelated to the septum, or whether it actually occurs in the substance of the septum after that structure has been formed. The consequences of each possibility will now be examined.

If the contents of the fold enter a preformed septum, then once the Müllerian ducts (which are the only contents of the folds meeting in the mid-line) have passed into the septum it is difficult to envisage what abnormal influence by the intestine could cause separation of their distal ends. Those portions which are not yet included in the septum, but which are due to fuse later in development, might conceivably be kept apart by bowel pressure. Such abnormalities as *uterus bicornis* could be explained in this way, but cases in which non-fusion of the lower ends of the ducts persisted to give, for example, a double uterus could not be so easily explained on such an assumption. If, on the other hand, the urogenital folds come together outside the septum, then they must develop on a plane ventral to it because the genital ducts must descend anterior to the septum in order to enter that part of the cloaca which will subsequently become the urogenital compartment. If, under these conditions, the Müllerian ducts and the folds in which they are contained are to be kept apart by the bowel, then it might be expected that, on occasion, the abnormal influence responsible for their separation would also prevent the urorectal shutter from falling completely posterior to them; in which case any malunion of the ducts whatsoever would be associated with the persistence of a communication between the distal bowel and urogenital compartment. Though abnormalities arising as a result of non-fusion of the Müllerian ducts are not uncommon, the coexistence of such a communication is very rare. The specimen described would appear to support such a conception, but, as will be shown, a more satisfactory explanation for the malformation is available. An examination of the observations supporting this theory, then, has produced evidence of a very conflicting nature. From the practical side the greatest defect in this theory is, as Keith has so ably pointed out, that it explains only a very limited number of abnormalities of the hind end, whilst by far the majority are totally unaccounted for

Frazer (1931) is of the opinion that the changes leading to the formation of the septum are produced by the rapid growth of the mesoderm at the hind end of the body, where the mesodermal bed containing the Wolffian duct "fuses with its fellow of the opposite side, and thus makes a transpelvic bar or bed of mesoderm, in which, a little distance from the middle line, the Wolffian duct runs downwards and inwards on each side". This cloacal

septum which has been accomplished in large part by the mesoderm in which the Wolffian duct is embedded, gradually divides the ventral cloaca into ventral and dorsal parts and is completed by the sixteen millimetre stage. Later, on page 453 of his "Manual of Embryology", he states that:

> The cloacal septum then is essentially a division of the original cavity, not affecting the added ventral portion. Forming behind the terminations of the ducts, it rolls in the walls of the cloaca on each side, making them into the ventral wall of the dorsal division. At the same time it extends its activities caudally. Hence the "septum" can be said to grow cranio-caudally and also from the sides, and to pass very near to the dorsal wall and far from the ventral wall. Moreover, in its formation it rolls in the wall on its ventral aspect to form the new dorsal wall for the ventral division, and is doubtless in this way responsible for the implantation of the Wolffian ducts on this new dorsal wall, between the ventral sinus and the dorsal rectum.

Descriptively there appears to be some discrepancy in these accounts, though it is evidently assumed that the transpelvic bar so closely associated with the Wolffian ducts is the same as the septum. Thus Frazer, in contradistinction to Felix, states that the septum is essentially the result of bilateral partitions meeting in the mid-line, and in this he is supported by Retterer (1890 and 1893), who also believes that two lateral mesodermic folds meet to constrict the original chamber into two separate compartments.

That the septum is not outlined by the approaching Wolffian ducts is easily seen when one considers the fate of the lower ends of the ducts. If these ducts were responsible for the formation of this septum—and this is difficult to conceive because they never become mid-line structures—then, as the septum grows down to divide the cloaca, the distal ends of the ducts, assuming they are responsible for the growth of the septum caudally, would also proceed distally with it; these would then ultimately open in the region of the cloacal membrane. That they do not do so is well seen by considering such a series as is shown in Figure 253 of Frazer's "Manual of Embryology", where, even with the passage downwards of the septum, the Wolffian ducts have not altered the relative positions of their openings into the ventral cloaca. The looping of the Wolffian ducts in the septum as described by Frazer (1935) appears to be the result and not the cause of the downgrowth. Moreover, in many abnormalities where the rectum enters the bladder and proximal portion of the urethra, as a result of incomplete formation of the septum, the Wolffian duct elements have undergone normal development. Consequently some other factor must be responsible for the formation of the septum and its growth below the point where the Wolffian ducts enter the ventral cloaca; that it is due to rapid growth of the mesoderm is obvious, but the cause remains an unspecified one. This conception of the manner of subdivision of the cloacal chamber also suffers from the same defect as the one previously outlined in that the abnormalities which are explained thereby are very limited indeed, while the cause of the septum is still essentially unaccounted for.

An endeavour will be made later to show that the factor responsible not only for the formation of the septum but also for its growth beyond the site of entry of the Wolffian ducts into the ventral cloaca is the development of the Müllerian ducts. It is certain that these ducts are bilateral, that they do fuse in the mid-line, and are the only structures which do so in both sexes; they also proliferate caudally to reach the surface in order to form the vaginal cord.

Sir Arthur Keith (1908 and 1933) and Professor F. Wood Jones (1904) have utilized the evidence provided from a study of malformations of the hind end to elucidate the various phases of normal development occurring in this region. According to Wood Jones the communication between the hind gut and cloaca decreases relatively in size, owing to increased growth

of the ventral and dorsal compartments of the cloaca, and is finally obliterated. The hind gut then develops caudally as a post-allantoic downgrowth, this development being dependent on the growth backwards of the hind end of the body. This downgrowth finally meets the ectodermal anal dimple and the membrane which remains between the two cavities subsequently ruptures and is absorbed. Though the majority of the anomalies in which a communication persists between hind gut and urogenital derivatives may be clearly and readily explained on the basis of his conclusions, the manner in which the narrow intercommunicating passage is finally obliterated is still unexplained.

Keith believes that as growth proceeds there is a sliding backwards of the hind gut along the cloaca, thereby reproducing the various phylogenetical milestones in its development. The arrangement of structures at the hind end of the body thus passes consecutively through amphibian, reptilian, monotreme, marsupial and higher mammalian stages. Arrest of normal development at any stage in these circumstances is believed to explain the various abnormalities found. This theory, however, does not explain why the gut migrates backwards and the mere statement that it is an example of ontogeny recapitulating phylogeny is hardly sufficient. Such reasoning as far as the genito-urinary system is concerned is fraught with danger. In various lower types it is not yet definitely known how much the allantois contributes to the formation of the bladder, and it is quite conceivable that the changes taking place at the hind end in such forms are in no way identical or comparable with those taking place in man, where the bladder is almost, if not entirely, a hind gut derivative. Moreover, this theory denies the formation of a special cloacal partition, and this is a view with which very few will be found to agree. Keith's reasoning, however, has certainly been most profitable in solving many problems concerned with maldevelopment of the hind end of the embryo.

From a survey of the literature it appears to be generally recognized that the primitive single cloacal chamber becomes subdivided at some stage during its development by a septum into urogenital and gut compartments, though Keith adopts a different interpretation when he writes that

> . . . in the evolution of mammals we see that the rectum migrates backwards until its vent or anus almost reaches the surface of the perineum, leaving the greater part of the cloaca as the urogenital sinus.

How and why the septum develops are points which do not appear to have received an adequate explanation. From observations made during the examination of this malformation, supported by additional evidence provided from a study of similar anomalies recorded in the literature and the arrangement of the genito-urinary system in certain lower mammalian forms, it is proposed to suggest that the urorectal septum, which ultimately completes the obliteration of the communication between ventral and dorsal cloacal compartments—a communication already narrowed down by relative growth changes—is formed by the fusion of the Müllerian ducts in the mid-line to form a uterus and proximal vagina. Subsequent differentiation in the perineal region is dependent on the development of a cord, downwards in the mid-line, which in the female is canalized to form the vagina, but which undergoes no further change in the male. The growth of the post-allantoic gut back to the perineum is closely associated with, but not the result of, downward growth of the vaginal cord; it is a manifestation of growth distally of the tail end of the embryo. In this way certain anomalies of the hind end which are totally unaccounted for by theories based on a study of pure embryology may now be satisfactorily explained.

Frazer and Felix believe, however, that the cloacal septum is completed at the sixteen millimetre stage which antedates the apposition of the Müllerian ducts, for the latter does not take place until the twenty millimetre stage, whilst their fusion is delayed until the twenty-one or twenty-eight millimetre stage. This observation, which requires confirmation, would dissociate the ducts from taking any part in the formation of the septum. Finality on this question, however, can be reached only when all the missing details of structural differentiation in this region have been provided by the embryologist. In the present state of our knowledge it is deemed permissible to present a new theory, provided, first, that it explains more than any previous theory, and, second, that it does not transgress any well established facts.

It seems reasonable to suppose that the only structure that will obliterate the mid-line communication between the gut and urogenital chamber will be one developing in the mid-line between those two cavities, and since the only structures appearing in that situation are Müllerian duct derivatives, the uterus and vagina, it may be adopted as a working hypothesis that these structures are responsible for the shearing off of the hind gut from the ventral cloaca. It is to be noted that Müllerian duct elements are developed in both sexes, even though they may be present in the male in an attenuated form. The suggestion is that, during development, the Müllerian ducts open into the floor of the intercommunicating passage, and that as the two ducts come together and fuse in the mid-line they shear off the communication. This process is diagrammatically shown in Figure V, A. However, so long as the ducts remain completely unfused, or only partially fused distally, the passage linking the two primitive cavities is not encroached on and remains patent, in which case the gut opens into the bladder between the unfused ducts. This condition is well illustrated in the specimen described, and represents the most primitive arrangement possible. It might be argued that it is possible for the Müllerian ducts to fuse completely without involving the intercommunicating channel, providing the duct and bowel orifices open into the cloaca as illustrated in Figure V, B—that is, providing the ducts open into the roof and not the floor of the cloaca. This, incidentally, is essentially the metatherian arrangement. It is important to observe, however, that when such is the arrangement the fused ducts open into the cloaca and not onto the surface, so that immediately the vagina is extended to reach the perineum the gut can no longer open into the cloaca. Thus with the appearance of a vagina which opens on the perineum the rectum is compelled to migrate further dorsally in order to gain a separate opening on the surface. It is well known that, so far as normal development is concerned, whenever the vagina opens on the perineum the rectum always opens posterior to it. It may therefore be concluded that, no matter what relationship the Müllerian ducts bear to the cloaca during development, the progressive differentiation of their derivatives is responsible for separating the hind gut and ventral cloaca.

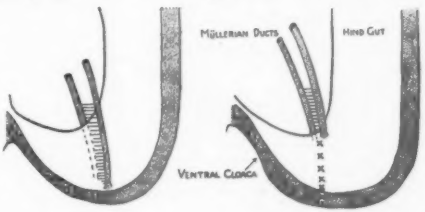


FIGURE V. Diagrammatic representation of the hind end to illustrate the manner in which the Müllerian ducts may terminate in the cloaca. The lines indicate where fusion between the ducts takes place, whilst the crosses indicate the direction taken by the developing vagina.

Phylogenetic studies, even though they do not offer conclusive proof in favour of the suggestion raised, are not incompatible with it. In the

amphibian condition the Wolffian and Müllerian ducts are unfused and open into the cloaca below the site of the bowel orifice (Figure VI, i). This is essentially, but with some modifications, the condition found in this specimen and that described in two of Keith's series. When, phylogenetically, the Müllerian ducts fuse, and they are the only ducts which do fuse in the mid-line phylogenetically and ontogenetically, one would expect the bowel to come to open into the cloaca below the fused ducts in order to escape constriction—that is, to adopt an arrangement similar to that represented in Figure VI, iii. In the monotremes, however, the two ducts remain widely open, and yet the bowel has migrated distal to the site of the duct openings into the urogenital sinus, which is that part of the cloaca receiving the outlet of the urinary system and the female genital ducts (Figure VI, ii). There is no fusion of ducts here to compel the bowel to migrate backward. It is quite possible, however, that in the monotremes the bladder is an allantoic derivative and so can claim little relationship with the hind gut; in such a case the ducts would necessarily open above the hind gut termination. It is also quite probable that the formation of the bladder from the hind gut is a higher mammalian feature.

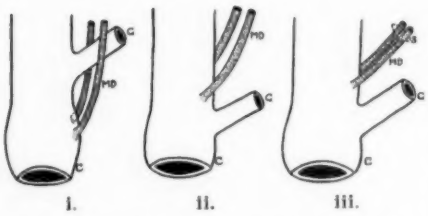


FIGURE VI. Series of diagrams to illustrate the variable relationship of the gut to the Müllerian ducts at their termination in the cloaca. i = amphibian; ii = monotreme; iii = marsupial; G = gut; C = cloaca; M.D. = Müllerian ducts; S = connective tissue or muscle sheath.

In marsupials, as in the monotremes, the gut opens into the urogenital sinus distal to the entry of the Müllerian ducts, but although the ducts remain separate they are closely bound together, side by side, in a common muscular or connective tissue sheath, which is mechanically the equivalent of fusion, whilst in some forms the septum between the distal ends of the ducts ruptures to form a single distal median vagina which breaks through to the urogenital sinus during parturition. The lateral vaginæ which are also characteristic features of the genital tract in the marsupials need not concern us since they are not mid-line structures and therefore do not affect this thesis.

The following is a brief survey of the variety of structural designs observed in marsupials. In *Myrmecobius fasciatus*, although the uterine bodies are separate, the two uterine necks are so closely bound together as to appear externally as a single tube. Each neck enters a median vagina below, which are again closely bound together to form a double median vagina which ends blindly below. This is the condition also found in the virgin *Perameles*, in which the distal common median vagina formed at birth to reach the urogenital sinus is repaired and has to be reformed at each succeeding parturition. In *Aerobates pygmæus* the two uterine necks, although separate, are closely bound together, whilst the double median vagina loses its septum posteriorly to give way to a short common median vagina. In *Tarsipes rostratus* and *Petaurus breviceps* the two uterine necks come together and are surrounded by a common muscular layer to open into a single short median vagina which is blind. After the first parturition, however, its communication with the urogenital sinus now remains permanently open. This is essentially the condition which is also found in the macropods. The final stages in this series of structural alterations is when the lateral vaginæ undergo phylogenetic senility and disappear, and the temporary median vagina becomes permanent to open ultimately on the perineum instead of into

the urogenital sinus. Thus in the marsupials there are bilateral structures which meet in the mid-line, and this may possibly explain the entry of the bowel into the cloaca distal to their openings. When the vagina comes to open on the perineum the rectal orifice will also be forced out of the cloaca onto the perineum. These phylogenetic changes are not cited as conclusive evidence supporting the theory suggested, for we are not yet in possession of sufficient data concerning the fate of the allantois in such animal types to compare the changes taking place there with those occurring in the ontogeny of man. They are merely included to indicate that a phylogenetic investigation reveals findings which are not incompatible with those necessary for the thesis under discussion.

The relationship of the *sinus pocularis* to the ejaculatory ducts in the male indicates that these parts of the Wolffian ducts always pass as far down the urethra as the Müllerian ducts. Any theory explaining the taking up of the Wolffian ducts into the urogenital sinus, so that the ureters and ejaculatory ducts open separately, must also explain why the ureter enters the bladder and the duct enters the urethra. Frazer (1935) has accounted for these changes in the following manner. "The first stage is effected by extension of the sinus along the duct, up to and just including the ureteric origin, the enclosed duct forming an intussusceptum (with the infolded sinus lining) which rapidly degenerates and disappears", so that the ureter comes to open directly into the sinus. "The second stage, following immediately, is marked by fixation of the duct by mesodermal condensation behind the sinus, with growth of this latter upwards in keeping with the growth of the infra-umbilical belly wall. The implanted end of the duct is thus turned up within the lining layer of the sinus, always at its lowest part, and the breaking down and disappearance of its front wall discovers the opening at the lowest level. In the meantime the upgrowth of the main cavity carries the ureteric levels farther and farther away." This stage is illustrated in Figure VII. Thus Frazer's explanation for the "looping" of the Wolffian ducts in the dorsal mesodermal sinus wall is that the process is due to growth changes which are in "keeping with the growth of the infra-umbilical belly wall". There is, however, an alternative explanation for this migration of the duct orifice down the sinus wall. Downgrowth of the distal ends of the Müllerian ducts in the dorsal wall of the sinus may quite possibly be responsible for dragging down the Wolffian ducts and thus outlining the loops so that, as before, one limb of the loop is thrown into immediate contact with part of the sinus wall, and subsequently degenerates with it exactly as Frazer has described. In such circumstances these loops would naturally pass as far caudally as the termination of the Müllerian ducts into the urogenital sinus in the male, and this would explain why the ejaculatory ducts finally open in the vicinity of the *sinus pocularis* in the prostatic urethra.

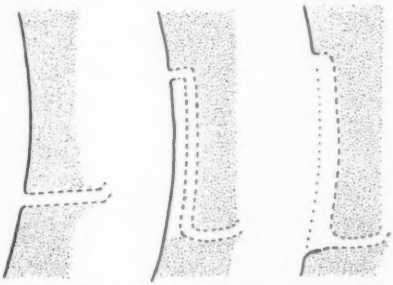


FIGURE VII. Series of diagrams to illustrate the manner in which the Wolffian duct comes to obtain a new opening into the urogenital sinus, and the manner in which the trigone of the bladder is outlined by the addition of part of the wall of the duct to the sinus. The taking up of the Wolffian duct so that the ureter comes to gain a separate opening into the bladder is not shown. The stippled area represents the mesodermal bed of the septum in which the ducts are passing to the sinus. The unshaded area in the second diagram represents that portion of the bed (that is, the dorsal wall of the sinus) which degenerates and is absorbed; its position is indicated in the third diagram by the dotted line. (Modified from Frazer.)

This theory will now be examined in the light of the abnormalities recorded in the literature. No previous case, so far as I am aware, has been described in which the rectum has entered the bladder in the female. This specimen is an example in which the Müllerian ducts have fused only over their most distal portions and so have failed to clip off the bowel, while the vaginal cord has either not formed or has formed and not become canalized, although histological examination has failed to reveal the latter possibility. The next cases are those in which the rectum enters the posterior fornix and proximal vagina. The explanation for such abnormalities is that the hind gut becomes linked with the fused Müllerian ducts and opens into that mass at the site of the shearing off process. When the shearing off process is complete, but the post-allantoic gut does not develop, then the rectum may be attached by a fibrous cord to any point over this vaginal area. The downgrowth of the post-allantoic gut is closely associated with, though not necessarily the result of, the development and downgrowth of the vaginal cord. Its development may be arrested at any point along the course of the vaginal cord, and this accounts for those cases in which the rectum either communicates with the distal vagina as far down as the navicular fossa of the vulva or is attached to it at any point over this area by a fibrous band. The bowel, however, may not grow distally and its connexion with the downward growing vaginal cord may be drawn out into a thin fibrous cord. Anteriorly the vaginal cord may canalize into the urethra or posteriorly into the rectum. In addition, the presence of a canalized vaginal cord in the female prevents the rectum from ever having a direct connexion with the urethra in that sex.

In males those cases in which the rectum enters the bladder or urethra proximal to the site of the *sinus pocularis* are due either to non-fusion of the Müllerian ducts or to fusion of only their most distal parts, so that the old communication between hind gut and ventral cloaca has not become clipped off. Attachment of the rectum to this region by a fibrous band is due to obliteration of the communication and failure of the post-allantoic gut to develop. Failure of the caudal development of the post-allantoic gut may be associated with communications between that gut and the urethra distal to the *sinus pocularis* or to fibrous connexions between the two. In these cases the ducts when fusing across the mid-line do not completely obliterate the communication, but leave it patent below, so that a communication persists between the rectum and urethra distal to the *sinus pocularis*.

The various stages in the development of the hind end of the embryo can by no means be considered as final. The evidence at this stage is so conflicting that dogmatism in the establishment of conclusions would be futile and ill-timed. Many of the statements made in connexion with this theory are certainly not given the status of definite facts; they are merely put forward as suggestions to which time may attach an element of truth.

SUMMARY.

1. An abnormality of the hind end of the body in a female is described.

2. The various theories relating to the development of this region and their relative values in explaining the malformations found, are discussed.

3. The suggestion is put forward that fusion of the Müllerian ducts is responsible for the separation of the hind gut from the ventral cloaca, and that further development of the hind end is associated with changes occurring in the distal ends of those ducts. The grounds on which such a suggestion is based are, firstly the conditions found in this specimen, and, secondly, its value in solving certain of the problems connected with the malformations recorded.

ACKNOWLEDGEMENT.

I wish to thank Mr. H. Marriott for the photographs accompanying this paper.

BIBLIOGRAPHY.

W. Felix: "The Development of the Urinogenital Organs" in Keibel and Mall's "Manual of Human Embryology", 1912.

J. Ernest Frazer: "A Manual of Embryology", 1931; "The Terminal Part of the Wolffian Duct", *The Journal of Anatomy*, Volume lxix, July, 1935, page 455.

Jas. P. Hill: "Contributions to the Morphology and Development of the Female Urogenital Organs in the Marsupialia", *Proceedings of the Linnean Society of New South Wales*, Part i, (Number 93), 1889, page 42, and Part iii (Number 99), 1900, page 519.

Arthur Keith: "Malformations of the Hind End of the Body", *The British Medical Journal*, Volume ii, 1908, page 1736 and page 1804; "Human Embryology and Morphology", Fifth Edition, 1933.

Augustus G. Pohlman: "The Development of the Cloaca in Human Embryos", *American Journal of Anatomy*, Volume xii, Number 1, July, 1911, page 1.

E. Retterer (1890): "*Sur l'origin et de l'évolution de la région anogénitale des mammifrères*", *Journal de l'anatomie et de la physiologie*, 1893-4; "*Mode de cloisonnement du cloaque chez le cobaye*", Bibliog. Anat., quoted by Pohlman (*loco citato*).

F. Tourneux (1893-4): "*Sur le mode de cloisonnement du cloaque*", etc. Bibliog. Anat., quoted by Pohlman (*loco citato*).

F. Wood Jones: "The Nature of the Malformations of the Rectum and Urogenital Passages", *The British Medical Journal*, Volume ii, 1904, page 1630.

DIVERTICULOSIS AND DIVERTICULITIS.[1]

By Hugh R. G. Poate,
Director of the Post-Graduate Surgical Unit, The Prince Henry Hospital, Sydney.

The selection of diverticulosis and diverticulitis as the subject for discussion this evening was influenced by two main factors: (*a*) its wide appeal to all branches of the profession and (*b*) the many difficulties in treatment of its complications.

Diverticulosis.

Prior to 1914 but scant attention seems to have been paid to the condition, and it was regarded rather as a curiosity, found occasionally in post-mortem examinations, although Virchow had drawn attention to the condition as far back as 1853. With the development of abdominal surgery a better understanding of the condition came about very gradually, but it did not receive any special attention until radiography came to be used more widely and its technique was perfected. For all practical purposes, then, our real knowledge of diverticulosis dates back only over the last twenty-five years or so.

The frequency of occurrence of diverticula in the alimentary tract is usually considered to occur in the following order: (i) colon, (ii) Meckel's diverticulum, (iii) duodenum, (iv) pharynx, (v) stomach, (vi) jejunum and ileum. It is not my purpose to discuss any of these areas other than the colon and only the acquired diverticula of this portion of the intestinal canal.

In the "Shorter Oxford Dictionary" a diverticulum is described as a "smaller side-branch of any cavity or passage", but Ian Fraser quotes the "Century Dictionary" and "Oxford Dictionary" as giving it a designation of "a way-side shelter or lodging", with from the context the underlying meaning that they are "houses of ill repute where trouble is apt to brew". From personal experience of the complications of this condition as affecting the colon, I can vouch for such an evil reputation, also their annoyance both to their owners and to the doctors called in to try to establish law and order.

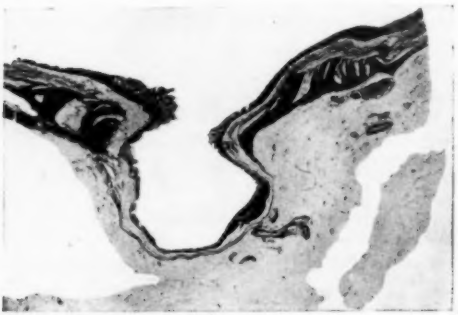


Figure I. Diverticulum opening into an *appendix epiploica*. Note the vessels at the apex. (Reproduced from article by H. Drummond, *The British Journal of Surgery*, Volume iv, 1916-1917, page 412.)

Structure of Diverticula.

Microscopic examination of acquired diverticula of the colon shows that they consist of a hernial protrusion of the mucosa and submucosa through the musculature of the bowel wall and are covered with a layer of peritoneum. Beneath the peritoneum there may be a variable amount of fat (see Figure I). Their constant relationship to the blood vessels of the bowel wall has been noted by many of the early writers, the earliest being Klebs in 1869, as also was their presence in conjunction with the *appendices epiploicæ*. Mention may be

[1] Read at the Queensland meeting of the Royal Australasian College of Surgeons on June 5, 1941. Accepted for publication on July 4, 1941.

made that these appendices are the terminations of the fat bands extending from the mesocolon around the bowel wall accompanying the blood vessels and develop at the point of entry of the arteries into the wall of the colon; this too is the usual point of development of diverticula (see Figure II). I recently performed laparotomy on a man who seemed to have diverticula connected with every one of the *appendices epiploicæ* on the iliac and pelvic portions of his colon. They were all swollen and thickened, as he had a chronic obstruction at the pelvi-rectal junction.

If diverticulitis has supervened, their appearance becomes greatly modified, and in severe cases necrosis may lead to perforation with either local or generalized peritonitis or local abscess. If the inflammatory reaction has been more chronic, a peridiverticulitis may result with adhesions to neighbouring structures, and there may develop a spreading proliferative reaction in the bowel wall which can mimic carcinoma very closely. Most surgeons have met with such cases and have resected areas in which later the thickening proved to be inflammatory, or in other cases have performed colostomy and given a bad prognosis, only to find that the patient kept on living.

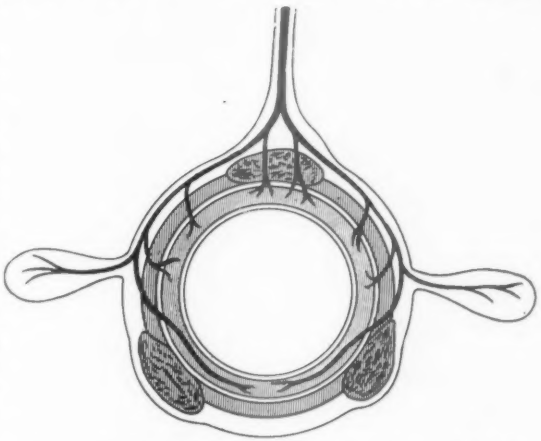


FIGURE II. Diagram to show muscle tissue and blood vessels of the pelvic colon. (Reproduced from article by H. Drummond, *The British Journal of Surgery*, Volume iv, 1916-1917, page 408.)

Telling in 1908 said of this condition:

> Of all the secondary results this proliferative inflammation is the most important, the most frequent and probably the most overlooked.

As long as the mouth of the diverticulum is open to the lumen of the bowel, inflammatory reaction is unlikely to ensue; but if the neck becomes narrowed and a stercolith forms, this may act as an irritant and cause necrosis of the mucosal lining, with consequent bacterial invasion of the walls, but frequently sclerosis is induced and the whole condition becomes quiescent.

Ætiology.

There has been a good deal of discussion regarding the ætiology, but as yet no general agreement has been reached. The position today in this respect remains much the same as it was when McGrath, of the Mayo Foundation, gave his masterly summing up of the condition in 1912. The fact that the majority of cases occur towards middle life and in the obese, has influenced opinion towards regarding constipation, irregularity of bowel action and straining at stool, intestinal flatulence and deposition of fat in the mesocolon or *appendices epiploicæ* as the major factors—in other words, that fatty deposition weakens the wall of the colon and that increased intracolonic pressure causes a herniation of the mucosa.

In straining at stool there is a forced raising of the intraabdominal pressure which must be applied evenly all round the colon, tending to force

any solid content in the lower part of the colon or rectum towards the anus, but unless flatus is present it is unlikely to be a factor of importance. Gaseous distension does play some part, although I have never seen or heard of diverticulosis associated with Hirschsprung's disease, with its immense dilatation and obstinate constipation, but probably the saving factor is the hypertrophy of the bowel wall in this condition. It is worthy of note that when complications do arise they are apt to occur at a relatively early age, whilst the uncomplicated cases are found chiefly in the elderly at post-mortem examinations or after X-ray examinations undertaken to assist in diagnosis of other ailments.

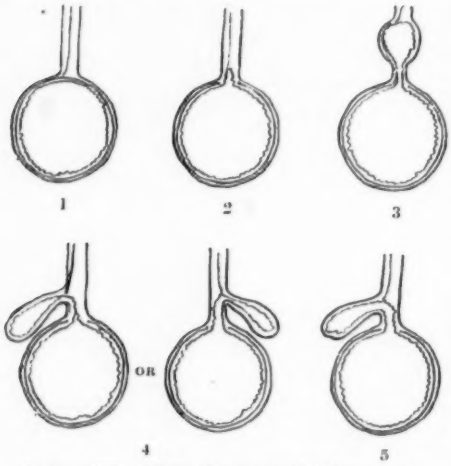


FIGURE III. Schematic representation of the development of a diverticulum. (Reproduced from article by Ian Fraser, *The British Journal of Surgery*, Volume xxi, 1933-1934, page 192.)

The condition does arise in comparatively young people, and a number of cases have been reported in persons under forty years of age; this makes it appear that degenerative changes in the wall of the colon with advancing years are not the whole story, and lends support to the view that there is an anatomical deficiency in the wall of the colon which is or can be a starting point for the development of a diverticulum.

In the small intestine it has been shown that the musculature of the bowel is weakest along the mesenteric border — an area which is unsupported by peritoneum, and that an added cause for herniation of the mucosa in this region is the deposition of fat which tends to weaken the muscle still further by a divarication of its fibres. The diverticulum forms at the point of entry of the artery through this weakened area, and takes shape as a protrusion of the mucosa which later tends to fall to one side. As it does this, the neck of the sac becomes drawn out and narrowed, giving rise to the typical pyriform shape of the usual diverticulum (see Figure III). A similar state of affairs holds in the colon, for diverticula are never seen apart from the entry point of the arteries. The relationship of these vessels is definite and constant, and is the only incontestable factor in the ætiology of the condition—a fact

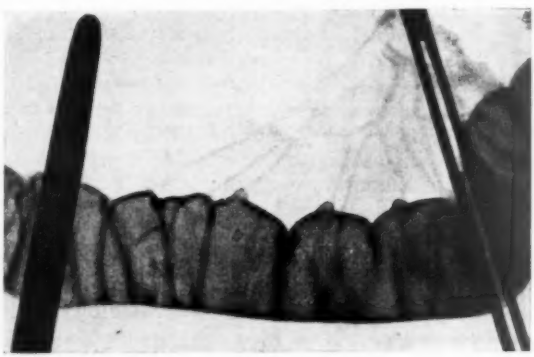


FIGURE IV. Artificially produced diverticula. Note each is related to an artery. Bismuth has been passing through the intestine prior to distension with oxygen. Forceps prevent the gas escaping. (Reproduced from article by Ian Fraser, *The British Journal of Surgery*, Volume xxi, 1933-1934, page 209.)

which was first pointed out by Hensemann in 1899. Ian Fraser has verified previous experimental work in the production of diverticula in the small intestine by distension alone, using a piece of fresh post-mortem gut. The distending medium was a thin bismuth mixture passed in under pressure. Diverticula are readily produced and consist of a herniation of the mucosa through the muscle at the point of entry of an artery (see Figure IV). He inclines to the view that jejunal diverticula are always the result of distension, while in the colon this factor is not so positive and muscle spasm may be of more importance. It is to be remembered that the artery has its accompanying vein and that anything which may cause venous distension is to be regarded as a factor in the ætiology of diverticulosis. The fact that in the colon diverticula are related to the *appendices epiploicæ* is because these structures contain the artery and their fatty content lifts the peritoneum, thus adding to the lowered resistance of an already weakened area. Gaseous distension of the colon is of common occurrence and would constitute a factor in the development of diverticula since it is usually associated with constipation.

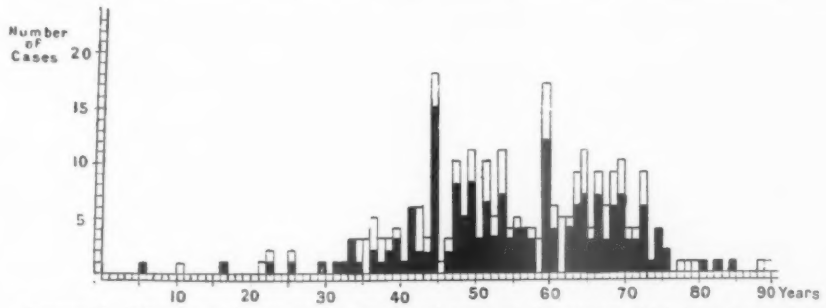


FIGURE V. Diagram to illustrate the number of cases of diverticula which have been met with at successive ages. The black areas represent male patients; the white areas represent female patients. The chart shows the greater incidence of the disease between the ages of forty-five and sixty. The greatest number of cases met with at forty-five, fifty and sixty is of interest. (Reproduced from article by Telling and Gruner, *The British Journal of Surgery*, Volume iv, 1916-1917, page 478.)

To summarize the factors in ætiology, one can say that the point of vascular penetration through the musculature of the bowel wall constitutes a point of weakness. General factors influencing the health of the individual or tending towards loss of muscle tone may increase the divarication of muscle fibres at this spot. Fatty infiltration may also play a part, as may chronic constipation or chronic spastic colitis, with either of which gaseous distension is of common occurrence. No single factor can explain the origin of acquired diverticula in the colon, and it is much more likely that the process results from a summation of these several factors.

Incidence.

Most textbooks state that diverticulosis is a condition found in persons above forty years of age, and regard it as one of the degenerative changes of advancing years. However, many cases are being reported in younger people and even in children (see Figures V and VI). Carl Bearse mentions in a recent article that he has operated on four patients under forty years of age for acute complications and that in one year he saw three other young people with diverticulosis. Judd and Pollock have operated on two patients under thirty years of age, and elsewhere in literature similar cases are continually being reported. It would appear therefore that the old ideas as to the age period for this condition must be revised. Rankin and Brown,

of the Mayo Clinic, reviewed a series of 592 cases of which 481 were clinical and 111 necropsies. Their figures are as shown in the accompanying table.

Group.	Number of Cases.	Decades.						
		20–29.	30–39.	40–49.	50–59.	60–69.	70–79.	80–89.
Clinical series ..	481	1	19	80	191	154	40	4
Necropsy series ..	111	1	—	6	30	41	31	3
Total	592	2	19	86	221	195	71	7
Percentage ..	..	0·03	3	15	37	32	10	1

Worthy of note is that over 3% were found in persons under forty years of age. Of 24,680 X-ray examinations of the colon, 5·67% revealed diverticula—a figure which should be high, as these are a selected series of cases in which investigation had been prompted by symptoms of some sort, yet in necropsies the incidence is 5·2%. It is generally concluded that the actual occurrence for all ages is less than 1%. The condition occurs rather more frequently in males than females, about 60% of sufferers being males. About 10% of patients with diverticula develop a subsequent diverticulitis.

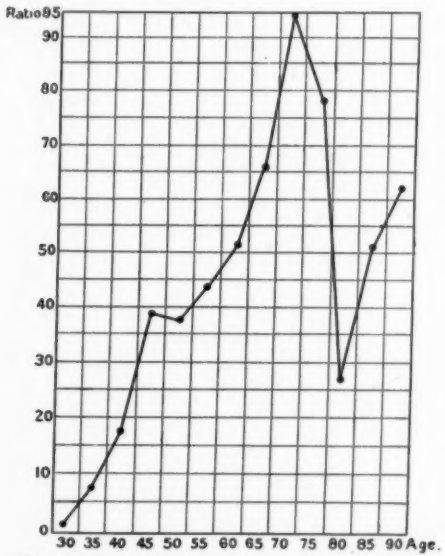


FIGURE VI. Diagram constructed from the same figures as Figure V, the necessary corrections being made to bring out a nearer approximation to the absolute frequency of the occurrence of diverticula in relation to the population living at each successive age. (Reproduced from article by Telling and Gruner, *The British Journal of Surgery*, Volume iv, 1916-1917, page 478.)

DIVERTICULITIS.

Inflammatory reaction in diverticula is brought about chiefly by obstruction due to a fæcolith causing pressure necrosis and allowing of bacterial invasion. The onset of diverticulitis seems to be more likely in the younger age periods and where the number of diverticula is few. At times it is either impossible or very difficult to demonstrate diverticula by X-ray examination, although clinical evidence is very strong and in some cases their presence can be demonstrated only at operation.

Bearse considers that the principal factors precipitating infection within a diverticulum include straining at stool, harsh cathartics, enemata administered with excessive pressure, and strenuous exertion, while foods which irritate the colon or increase its bacterial content may be contributory causes. Diverticulitis may lead to other complications, and it seems that perforation, abscess formation or peritonitis is more likely to occur in the younger people, but chronic obstructive lesions are more likely to occur in the older age groups, being due to a low-grade infection permeating

the wall of the diverticulum and extending along and through the wall of the colon. The bowel wall may become grossly thickened and simulate a carcinoma very closely.

Symptoms.

Diverticulosis in itself causes no symptoms, but if associated with a spastic colon or other form of chronic colitis, the usual chain of symptoms incident to these conditions is present. With the onset of diverticulitis, however, the clinical picture changes and varies according to the acuteness of the inflammation, as to whether perforation of the diverticulum occurs or what nearby viscera are involved.

In people over thirty-five years of age pain in the left iliac fossa is very commonly due to diverticulitis in one form or another. Constipation is usual, but attacks of diarrhœa or the passage of mucus and even of small amounts of blood may occur. With chronic diverticulitis the pain is usually intermittent, is common towards the end of the day and is seldom constant. In character the pain is likened to a dull gnawing ache with occasional sharp spasms, and there may be some general abdominal discomfort. If an acute infection is present, then pain may be very sharp and be accompanied by nausea, vomiting, feverishness, tenderness and muscular resistance. In any doubtful case a leucocyte count is very helpful and may be of prognostic import. If any degree of local peritonitis occurs, or if there is an acute perforation, symptoms and signs in the left side of the lower part of the abdomen may develop on parallel lines to those of a ruptured appendicitis.

Varieties and Complications.

The best classification of diverticulitis seems to be that of Rankin and Brown and is as follows:

1. Acute.
2. Chronic.
3. Complicated—
   - (*a*) Abscess formation.
   - (*b*) Fistula—
     - (i) External.
     - (ii) Internal: vesico-colic, entero-colic.
     - (iii) Multiple.
   - (*c*) Associated with carcinoma.

I shall now consider these different varieties and discuss briefly the salient points of illustrative cases.

Acute Diverticulitis.

Acute diverticulitis is most commonly the result of mucosal erosion and infection in a single diverticulum due to the presence of a fæcolith in its lumen. Until such time as the peritoneum becomes involved, it may produce no symptoms, except some slight malaise. Pain is then felt usually in the left iliac fossa, and it varies according to the acuteness and extent of the inflammatory process induced. If it is a localized and low-grade infection, pain will be referred to as a constant dull ache and there may be very little constitutional disturbance, but there is generally a point of tenderness. This infection in a diverticulum is of very common occurrence, and is probably the most common cause of left lower abdominal pain in persons over forty years of age. As a rule the pain subsides after a few days, but is liable to recur at irregular intervals.

If the infection is more severe, a local peritonitis will result, with acute pain, tenderness, muscle guarding, some rise in temperature and a

leucocytosis. Symptoms may persist for several days. Recurrence is apt to occur, especially after indiscretions in diet.

A Jewess, thirty-six years of age, was seen by me some five years ago with symptoms of this nature which subsided after some ten days. Later diagnosis was confirmed by X-ray examination and she was cautioned as to diet *et cetera*. However, she will not keep to instructions and every six to twelve months she has a recurrence of symptoms.

Another elderly woman, of over sixty years, was first seen in a very severe attack which took several weeks to subside, but every now and then she throws all restrictions aside and insists on having a party, as "otherwise life would not be worth living"—with the inevitable result of a flare-up.

The fulminating type of acute diverticulitis may be alarming in its severity, and unless there is some positive means of arriving at a diagnosis, laparotomy may be undertaken under an erroneous diagnosis.

Only three months ago I was called to see a man of forty-two years of age with a history of a chronic duodenal ulcer confirmed by X-ray examination, during which it was noted that he had extensive diverticulosis of the pelvic colon. An acute abdominal crisis developed early one morning following on a dinner party. It was thought that he had a ruptured ulcer, but the pain was referred to the lower part of the abdomen, which was rigid. He had a temperature of 103° F. and a pulse rate of 130. Tenderness was centred over the iliac colon and was very acute. I diagnosed an acute diverticulitis and advised removal of the patient to hospital for treatment. His own doctor urged laparotomy, and, when I declined, asked for another opinion, which favoured delay. He had a leucocytosis of some 18,000, but two days later it was 12,000, and he gradually improved, but on the twelfth day had another similar attack. This caused us great concern, but relying on the protective adhesions which he must have developed, we again adopted conservative measures and he recovered slowly and so far is sticking strictly to instructions.

In this type of case natural cure is the usual result, due to shutting off of the area by adhesions and their fibrosis. One frequently finds adhesions about the pelvic colon in performing laparotomy and this is the explanation of their occurrence.

Chronic Diverticulitis.

Chronic diverticulitis has also been termed sigmoiditis and of recent years peridiverticulitis. Both of these are bad terms, as the condition can occur anywhere in the colon and is a good deal more than an inflammatory reaction around a diverticulum, although it commences there. This is the condition which may simulate carcinoma so closely that even with the help of an expert radiologist it may be difficult to make a diagnosis, and on occasions even after laparotomy many mistakes have been made.

The inflammatory reaction spreads along and around the wall of the colon, involving all layers. It is not uncommon to find a mass ten to twelve centimetres in length and five to eight centimetres in diameter. Symptoms are often slight and misleading until a chronic obstruction develops which may on occasions become acute. This tumefaction is a feature of chronic diverticulitis, and it takes much experience and clinical acumen to be certain of diagnosis.

Some fifteen years ago a well-known man was found to have a mass in his left iliac region. At operation performed by one of our most distinguished surgeons, now in other realms, an attempt was made to separate it, but as infiltration seemed so widespread the abdomen was closed and a hopeless prognosis was given. The patient was very ill after this and finally developed an abscess, which was drained. He made a rapid recovery, but was left with a fæcal fistula which he still cherishes, and is now a hostile critic of surgeons in general and refuses any attempt at cure.

A few years ago I was asked to see a woman of fifty years who had been operated on three weeks previously by a gynæcologist for a mass in the left side of the pelvis involving the tube and ovary, which were glued to the colon. The tubo-ovarian mass was separated with difficulty and removed. The patient had a very stormy time and a week later developed a fæcal fistula through the incision. Normal bowel action ceased and all fæces came through the abdominal wall. By the end of the third

week she was in a low delirium and in a pitiable state. Gas-gangrene antitoxin was given and she made a miraculous response, so that a few days later transverse colostomy was carried out. To make a long story short, she eventually had a resection of sinus and involved colon and has been well ever since.

The lesson to be learned is never to tamper with this condition if found at laparotomy. If it can be done, wrap omentum around it and fix it with a couple of stitches, and then perform a transverse colostomy. With care in treatment and patience the mass may, and usually does, resolve without stenosis, and in some six months the colostomy may be closed. If stenosis is present, then resection can be safely undertaken after some months. Never perform immediate resection, as you are almost certain to produce a spreading cellulitis or peritonitis leading to death of your patient. We can all make mistakes, but we should never make the same mistake twice. I learned my lesson many years ago in resecting what I thought was a carcinoma of the pelvic colon, but what proved to be a chronic diverticulitis, and the patient died. Since then I have had a great respect for this condition.

Complicated Diverticulitis.

Complicated diverticulitis would perhaps be described better as the complications of diverticulitis.

Abscess Formation.—Abscess formation is a result either of perforation of an acute diverticulitis from a gangrenous patch in its wall or of the permeation of organisms resulting in fat necrosis and secondary abscess formation. The former condition occurs following a severe attack of acute diverticulitis, and is characterized by a prolongation of the illness, whereas the latter may occur after an apparently mild attack of diverticulitis and may cause little in the way of symptoms other than a complaint of malaise and debility. As an example of this chronic abscess I may mention two illustrative cases.

The first was in a woman of middle age who had been "off colour" for a couple of months and then noticed a soreness above the pubes. A hard mass was felt extending into the left iliac region and *per vaginam* could be felt in the left side of the pelvis. An incision was made and an extraperitoneal inflammatory mass was revealed which on needling yielded pus some two centimetres from the surface. This was drained and the discharge of flatus and a little fæces confirmed the diagnosis. The condition cleared up completely in some six weeks.

The second case was that of a man of over sixty years who was known to have extensive diverticulosis with occasional mild attacks of diverticulitis. He had been operated on five years previously for a gangrenous gall-bladder, a condition, by the way, which in my experience is commonly associated with diverticulitis. He had been "feeling cheap" for a month, had a furred tongue, and complained of anorexia *et cetera*. He had not had any diverticulitis for at least six months previously, and after this attack the X-ray appearances after an opaque enema showed no difference from those noted at X-ray examination a year previously. Nothing positive was found on examination, but three days later he developed typical signs and symptoms of a very acute attack of appendicitis. Operation was undertaken within eight hours of the onset, and a very acutely inflamed and gangrenous appendix was located along the pelvic brim; when it was separated pus welled up from the pelvis. The appendix was removed, but showed no perforation, and it was thought that the patient must have had some transudation of organisms from a symptomless appendicitis of some weeks' duration which had caused the abscess and that the recent acute symptoms showed out only when the appendiceal artery became thrombosed. A few days later, when fæces and flatus came away, the diagnosis was altered to that of a perforated diverticulum of the pelvic colon with a pelvic abscess (high up and not palpable *per rectum*) which ultimately involved the appendix. This man died some fifteen months later with carcinoma of the colon and is the only case that I have known in which there has been a direct relationship between these two conditions.

It may be mentioned here that a number of cases of metastatic abscesses have been reported following on a diverticular abscess, as also has suppurative

pylephlebitis. One case of multiple lung abscesses and a brain abscess have been reported. Subphrenic abscess may also occur, and I remember seeing one in a woman who had been operated on by a gynæcologist and in whom an acute diverticulitis was finally diagnosed after much handling. It appeared clear in her case that pus had tracked up lateral to the descending colon and had formed a huge left subphrenic abscess.

Fistulæ.—As already noted in the classification of diverticulitis, fistulæ may be external, internal or multiple. The most common type of internal fistula is the vesico-colic, and Arnheim lists a group of 1,516 cases of diverticulitis in which the incidence was 8%. In the majority of such cases the fistula develops low down on the posterior wall of the bladder and is thus in a very difficult position in regard to surgical approach for a radical cure. This should be undertaken only when all acute symptoms have subsided and after the bladder is as clean as it can be made and the renal function is normal. The only way to ensure this as well as the success of your operation is to perform a colostomy as early as possible to suspend the function of the lower part of the colon and to allow of its irrigation.

This condition is rare with carcinoma; in fact, it is so unusual that one can almost take it for granted that vesico-colic fistula is always secondary to diverticulitis.

External fistulæ are common and are usually due to the mishandling of an acute diverticulitis wrongly diagnosed and operated on as tubal inflammation. It may occur after incision of a diverticular abscess either through the abdominal wall or through the pouch of Douglas. I have already referred to this complication in some of the cases mentioned. Entero-colic fistulæ are not common, but are sometimes met with in the course of a laparotomy for some other condition. The patient who does develop one is lucky, as natural cure of the diverticulitis results and the fistula as such is symptomless.

Carcinoma.—We now come to the question of malignant disease in relation to diverticulosis or associated with diverticulitis. I have already instanced the only case that I have met with in which carcinoma did supervene shortly after an acute diverticulitis, but in general it seems agreed that the frequency of carcinoma in people suffering from this condition is no greater than it is in the general population.

Arnheim states that diverticulitis of the colon was associated with carcinoma in only 19 of a group of 1,600 patients operated on for carcinoma of the colon—that is, just over 1%.

Carcinoma of the pelvic colon is a very common condition and so is diverticulosis, yet Dr. Sear tells me that he does not remember a case in which he has been able to demonstrate the two conditions by X-ray examination.

Strangely enough, I have two patients at the present time with the dual condition.

One is in a man of eighty-three years who had a symptomless diverticulosis with a carcinoma at the pelvi-rectal junction; but excision was negatived as the growth was too advanced.

The other is in a man of fifty-three years who had a carcinoma at the lower end of the pelvic colon which was causing chronic obstruction. His pelvic colon was very long and was studded with dozens of diverticula which were involved with the bowel in a subacute inflammatory condition along with the chronic obstruction. These diverticula were two to three centimetres in length and stood out at right angles to the colon, presenting an extraordinary appearance, the like of which I have never seen before. It was difficult to find an area in which to perform a colostomy. The patient is very well at present and we hope soon to be able to perform a resection with safety.[1]

[1] At this stage a number of lantern slides lent by Dr. Sear and Dr. Anderson Stuart were shown illustrating various types of the disease and some of the difficulties in the X-ray diagnosis.

Treatment.

Treatment will be considered under two headings: medical and surgical.

Medical Treatment.

Diverticulosis.—It is to be remembered that diverticulosis is a common condition occurring in about 1% of all persons over forty years of age, but of these only 5% to 15% (authorities vary as to the percentage) develop any sign of diverticulitis. It is wise, if and when the condition is discovered, to point out to the patient that it is not a cause for alarm, but that it is as well to adopt some elementary dietary control. Avoid foods liable to leave indigestible residue or to increase decomposition within the colon—for example: skins or pips of fruit either fresh, cooked or in jams; nuts, cabbage, the stalk of cauliflower, turnips of any variety, celery, radishes, watercress, beetroot, preserved fish or meat of any kind, sauces, pickles, curry or other condiments; a minimum of red meats should be taken and all vegetables should be minced. Avoid cathartics and be careful with enemata. Take small doses of liquid paraffin daily, the best amount being one teaspoon morning and evening, as large doses tend to leak through the anus and may incline the patient to discontinue its use. If there is any tendency to spasticity of the colon or any degree of colitis, however mild, strict adherence to these measures must be enjoined. If the patient is over-weight, further dietary restrictions may be advisable. There is no drug likely to influence the condition, although belladonna still has its advocates if there is any spastic condition of the colon, but I prefer regular and prolonged use of vitamin B_1 in such cases. Intestinal antiseptics, so-called, have had their vogue, but it seems generally recognized that little if any antiseptic action from such drugs is possible, and recent work on the action of antiseptics rather negatives any such effect of these drugs. The patient should be warned that if any great change occurs in the bowel habit, or if mucus with or without blood is passed, he should report at once and have a further X-ray examination carried out.

Diverticulitis.—With the onset of diverticulitis the whole clinical condition alters, as does the treatment once any subacute or acute inflammatory condition sets in. The minor degree, without general symptoms and with only a dull ache in the left iliac fossa, is best treated by intensification of the dietary control and perhaps by daily colonic lavage with permanganate of potash solution, one in 1,000, or even stronger. When pain is a feature and there is any constitutional disturbance, complete rest in bed is imperative and fluids only are to be given by mouth. A full course of sulphanilamide is most helpful, and in very acute conditions I advise that this should be supplemented with gas gangrene antitoxin given by intramuscular injection in full doses. Warm olive oil injection along with the application of heat over the affected area of the abdomen gives relief. Sedatives in sufficient quantity to relieve pain and give rest are indicated, and the Fowler position should be adopted in order to try to limit the inflammatory spread to the lower part of the abdomen. Olive oil may also be given by mouth as well as small doses of liquid paraffin, and if aperients are required the compound liquorice powder is very useful. Avoid strong purgatives, and especially calomel and salines. The patient should be warned as to the necessity of adhering strictly to diet for all time, as indiscretions in this respect are very apt to be followed by acute attacks.

It is unwise ever to conduct a sigmoidoscopic examination in acute or subacute conditions as the bowel wall and the walls of the diverticula are inflamed and rigid. Any undue pressure by the instrument or the inflation of the bowel may cause a rupture, and I know of two cases in which the sigmoidoscope has been pushed through the bowel wall with fatal results.

It is wise in any case at all in which a sigmoidoscopic examination is carried out, not to use a general anæsthetic, to desist if pain is caused, and to use minimal inflation of the colon.

I would advise investigation of the gall-bladder in all cases of acute diverticulitis, as if there is any chronic cholecystitis or if biliary calculi are present, the onset of an empyema of the gall-bladder is by no means an uncommon complication.

Surgical Treatment.

Surgical treatment is indicated only in the presence of the complications of diverticulitis as already outlined.

If by some mischance the abdomen has been opened and a localized acute diverticulitis is found, the greatest care in its handling must be exercised and the only surgical manœuvre permissible is to wrap the affected segment of colon around with omentum if it is available and to fix it by a few sutures to the peritoneum in the iliac fossa or to the mesocolon and not to the bowel wall. This will limit the infective process and if an abscess does form it will be localized. If it is possible, lift the affected area up from the pelvis and fix it in the iliac fossa. On no account should an inflamed diverticulum be resected as any suturing of the wall of the colon will assuredly break down and general peritonitis will ensue or at best a fæcal fistula will result. The less handling that the colon in a case of acute diverticulitis receives, the better the chance for the patient. I would issue a special word of warning in regard to any gynæcological procedures, as the most tragic cases I have seen have always resulted from either an attempt to free the colon from the uterus or its adnexa or from the removal of tube or ovary involved in the inflammatory mass.

Early surgical intervention in acute diverticulitis where resection either of the diverticula chiefly concerned or of a segment of the bowel itself is carried out, is attended with a mortality of 60% or over, whereas with conservative measures the mortality is only some 15%. Arnheim gives a mortality rate of 62% in a series of cases with surgical complications. P. W. Brown, of Rochester, in discussing 191 cases in which various surgical procedures were used, reports a mortality rate of 18·3% when death ensued after operation or from related conditions and a further 14·1% when death was caused by unrelated conditions; only 44% of patients could be classed as cured; in the remaining cases symptoms persisted over a period of years.

In association with D. M. Marcley, Brown found that surgical treatment was required in 9% of 386 cases of acute diverticulitis, but in commenting on 277 of these cases treated medically he expressed the opinion that the only conclusion warranted was that patients who had an uncomplicated diverticulitis and who followed a medical regimen seldom required surgical treatment.

Attention has been drawn to the greater frequency of surgical complications in patients under sixty years of age and in Arnheim's series 35% were under forty years, but his is a smaller series than Brown's. It is of interest to note that he lists the frequency of complications of acute diverticulitis as follows: peridiverticulitis, 20%; abscess, 19%; peritonitis, 12%; and vesico-colic fistula, 8%.

Brown states that in about 16% of cases symptoms develop rapidly and operation must be performed within a month after the first symptoms appear. In fully 50% of cases operation has to be performed within the first year and in the remaining cases the patients carry along from bad to worse for one to several years before operation is undertaken.

I have not been able to dissect the figures given for mortality rates according to whether the lesion has been acute, chronic or complicated.

So far my remarks as to treatment have been confined to the acute conditions before abscess formation has taken place or complications other than a local peritonitis have occurred.

If perforation has occurred or if a gangrenous diverticulitis is present, establish local drainage and perform an immediate transverse colostomy.

With abscess present, it is wise to delay surgical intervention until the abscess has become securely walled off and has come within easy reach of the surface. Again the minimum of operative interference is the wise course—simply put in a tube and drain the abscess.

When there is any degree of chronic diverticulitis producing obstruction which cannot be relieved by medical treatment, the wise course is to perform a transverse colostomy so as completely to defunction the involved area of colon. Let the patient wait for some months, during which daily lavage of the lower part of the colon and rectum is carried out. This often results in absorption of the inflammatory mass and restoration of the lumen of the bowel, a condition easy to judge by X-ray examination. If stenosis persists, resection can be performed. Once the lumen of the colon has been restored, the closure of the colostomy can be undertaken with safety.

If fistulæ, either external or internal, are present, it is imperative before carrying out any attempt at cure to establish a transverse colostomy and to cleanse the lower bowel for some months.

It may be found that the fistulous track connects up with a grossly contracted area of colon and that resection of this stenosed area is necessary. Such a procedure calls for the highest surgical technique in which one cannot afford to take risks. Play safe and do not yield to the importunities of patients or their friends to "do something" until you are satisfied that both the general state of the individual and the local bowel condition justify a major surgical attack on the involved area. In such cases a blood transfusion may be required and steps should be taken to have it ready at the time of operation.

Prognosis.

P. W. Brown, of the Mayo Clinic, in his most recent article states that he continues to be perplexed by the fact that diverticulitis develops in only a small proportion of patients who have diverticulosis, and he thinks that the number who experience sufficient trouble to seek medical advice is not more than 10% of the total number with diverticulosis. He also remarks that a peculiar predestination seems to be present in cases of diverticulitis; some patients may have no symptoms, in others they may be moderate, severe or even fulminating, so that an obvious major abdominal catastrophe may be the first sign of the trouble.

As far as diverticulosis is concerned, it seems fairly evident that it is a condition which, if found, is not greatly to be feared, particularly after the sixty year period, as only a small number of surgical complications occur in the later years, the majority of them being the chronic infiltrating condition which leads to stenosis and may be mistaken for carcinoma. Vesico-colic fistula is usually met with in the elderly. The younger the individual, the greater the risk of an acute surgical emergency and also the greater the mortality. Once symptoms have become evident the patient should be warned of the necessity for a lifetime dietary control, and if patients will adhere to this advice it seems that very few will ever develop diverticulitis of sufficient severity to demand surgical measures.

It is interesting to note that although the frequency of diverticulitis is very slightly more in the male than in the female, the incidence of surgical complications is nearly double in the male. If acute complications arise, it is best to postpone operation to as late a stage as possible and to disturb the *status quo* as little as possible. It is far safer to tide the patient over an acute emergency and to leave any curative surgical procedures until such time as they can be carried out deliberately and under the best possible conditions. I feel sure by such practice the mortality rate can be lowered greatly.

SUMMARY.

1. Diverticula of the colon are of common occurrence and, being narrow-necked thin-walled herniæ of mucous membrane covered by peritoneum beneath which there may be variable amounts of fat, they are liable to cause various troubles if inflammation sets in.

2. The ætiology of the condition is probably a summation of several factors acting on an area of bowel wall where some muscular weakness has developed in relation to the site of entry of the blood vessels.

3. The principal factors precipitating acute diverticulitis include straining at stool, the use of harsh cathartics, enemata given with excessive pressure and strenuous exertion.

4. The most common cause of pain in the left iliac fossa in patients over forty years of age is diverticulitis of one form or another.

5. The younger the patients, the more likely are they to develop acute surgical complications which have a high mortality if subjected to immediate operation.

6. The main thing to remember in dealing with acute diverticulitis is to display a watchful masterly inactivity, and if any surgical interference is demanded, to do as little as possible.

7. The incidence of carcinoma with diverticulitis is incidental rather than actual, but the chronic sclerosing diverticulitis may be difficult to differentiate clinically from carcinoma.

BIBLIOGRAPHY.

H. Drummond: "Sacculi of the Intestine, with Special Reference to their Relations to the Blood Vessels of the Bowel Wall", *The British Journal of Surgery*, Volume iv, 1916-1917, page 407.

W. H. Maxwell Telling and O. C. Gruner: "Acquired Diverticula, Diverticulitis, and Peridiverticulitis of the Large Intestine", *The British Journal of Surgery*, Volume iv, 1916-1917, page 468.

F. Gordon Bell: "Diverticulitis", "Transactions of the Australasian Medical Congress (British Medical Association), Third Session", 1929, page 540.

B. F. McGrath: "Intestinal Diverticula: Their Etiology and Pathogenesis", *Surgery, Gynecology and Obstetrics*, Volume xv, October, 1912, page 429.

Carl Bearse: "Acute Perforative Diverticulitis of the Colon in Young Persons", *The Journal of the American Medical Association*, Volume cxiii, Number 4, 1939, page 1720; "Diverticulosis and Diverticulitis of the Colon, with Special Reference to Patients under 40", *Review of Gastroenterology*, Volume vii, July-August, 1940, page 318.

F. W. Rankin and P. W. Brown: "Diverticulitis of the Colon", *Surgery, Gynecology and Obstetrics*, Volume l, May, 1930, page 836.

C. Bickerton Blackburn: "Diverticulosis and Diverticulitis", *The Medical Journal of Australia*, Volume ii, 1938, page 405.

H. R. Sear: "Diverticulosis and Diverticulitis from the Radiological Point of View", *The Medical Journal of Australia*, Volume ii, 1938, page 409.

P. W. Brown and D. M. Marcley: "Prognosis of Diverticulitis and Diverticulosis of the Colon", *The Journal of the American Medical Association*, Volume cix, October 23, 1937, page 1328.

P. W. Brown: "The Treatment and Prognosis of Diverticulitis of the Colon", *The American Journal of Surgery*, Volume xlvi, 1939, page 162.

E. E. Arnheim: "Diverticulitis of the Colon, with Special Reference to the Surgical Complications", *Annals of Surgery*, Volume cxii, September, 1940, page 352.

Surgical Technique.

INSTRUMENTAL METHODS OF TYING KNOTS IN INACCESSIBLE REGIONS.[1]

By A. C. McEachern,
Adelaide.

In general surgery it must be extremely seldom that it is impossible to apply a ligature in the ordinary way because of inaccessibility. Be that as it may, it would appear from the number of special forceps illustrated in catalogues that there must have been occasions upon which the need has been so great that these appliances have been designed.

One such occasion, which would serve as a possible example, is the ligation of the cystic duct and artery in an exceptionally stout subject where the point of ligature

Figure I. Ligature jigger for tying reef knots.

is reached with the index finger only at the cost of some discomfort to the surgeon. In such a case it would seem that a sure and comfortable and accustomed method of instrumental ligation might be safer and more satisfactory than the ordinary knot.

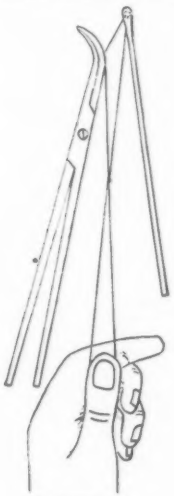


Figure II.

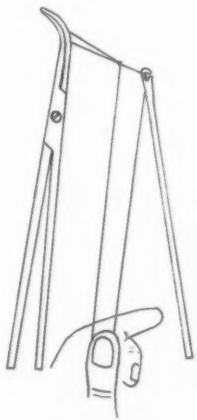


Figure III.

Figure IV.

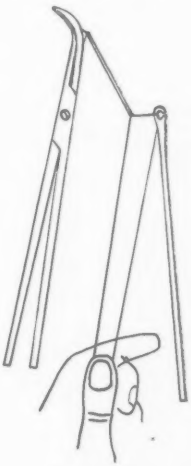


Figure V.

Figure II: The ligature is placed round the forceps. Figure III: The jigger is moved to commence the knot. Figure IV: The first part of the reef knot is tightened. Figure V: The second part of the knot is begun.

The simple instrument which is here illustrated (Figure I) merely replaces the embarrassed index finger, and retains a good deal of the finger's accuracy. The design of the instrument is not of great moment, but two points are of very considerable importance: there must be no sharp edge to damage the catgut, and no sharp point which might cause damage if the catgut broke. The actual method of tying the knot is clearly indicated in Figures II, III, IV and V.

[1] Accepted for publication on January 23, 1941.

It has been interesting to observe that the procedure has been adopted by a number of one's colleagues for tying vessels in the tonsil fossa.

The second method of knot tying (Figures VI, VII, VIII and IX) is commonly used by throat surgeons, but is not so universally known and used as its great efficacy

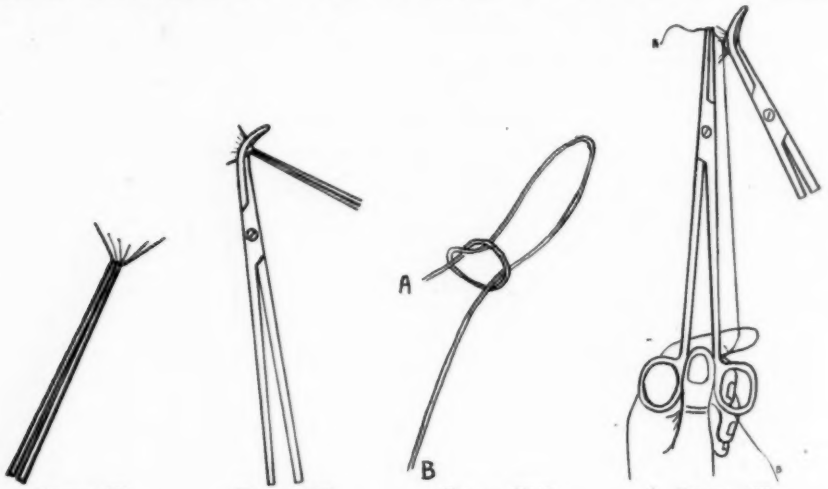


FIGURE VI. FIGURE VII. FIGURE VIII. FIGURE IX.

Figure VI: The bleeding point picked up with Waugh's forceps. Figure VII: Waugh's forceps replaced by curved artery forceps. Figure VIII: A simple loop knot with one short end. Figure IX: Straight artery forceps applied to short end, ligature tightened with left hand while artery forceps removed with the right.

and convenience would warrant. Theoretically the use of a slip knot would seem unsound, but in actual practice it unexpectedly "stays put". In several hundred tonsillectomies performed by a number of surgeons only one instance can be recalled in which it failed; and that was in a heavily built male who became temporarily maniacal while recovering from the anæsthetic.

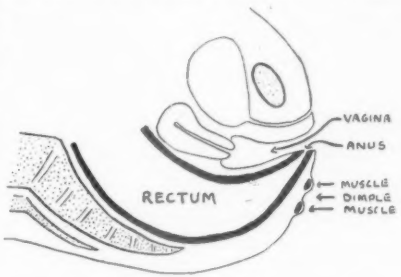
Case Reports.

A CASE OF CONGENITAL ECTOPIC ANUS TREATED BY OPERATION.[1]

By Douglas Robb,
Auckland.

Edna N., a girl of five years, was referred by Dr. S. Wallis, of Rotorua, on account of a congenital malformation of the anus with incontinence. During her first year she had been treated by Dr. Wallis for talipes with a good result. Her parents were Dalmatians, who were aware of no consanguinity in recent generations, and there was one sister nearly three years older. This sister had a series of congenital deformities, but was in good health and had developed well; both hands simulated amputation at metacarpo-phalangeal joints, and had rudimentary thumbs and tiny digits. All the toes were short, with partial syndactyly, more marked on the left than on the right side. In the mouth there was a deficiency in the centre of the upper alveolus, and several teeth were absent. The upper lip was indrawn as a result of this. There was a very short frenum to the tongue.

On examination under anæsthesia the rectum was found to open into the posterior aspect of the vagina just inside the vulva by a rigid opening slightly less than a centimetre across (Figure I). The vulva and vagina elsewhere were normal. Posterior to the opening described, in the position of a normal anus a subcutaneous ring of muscle could be seen and felt, with a shallow pit in the centre. It was decided to try to transplant the opening into the normal position in the hope of (*a*) rendering the vagina normal, (*b*) abolishing the anal atresia and (*c*) restoring a degree at least of continence.



CASE OF CONGENITAL ECTOPIC ANUS
SHOWING CONDITION BEFORE OPERATION.

Figure I.

The first operation consisted in a double-barrelled colostomy in the sigmoid colon through the left rectus muscle, in order to make the distal colon functionless, after the manner of Devine. The whole pelvis was then found to be filled with a column of inspissated fæces, doubtless the result of the narrow anal orifice. Energetic efforts to clean out this mass followed, and in five weeks' time the main operation was carried out.

The second operation began with a mobilization of the lower part of the rectum through a mid-line incision behind the anal dimple. The rectum was tremendously hypertrophied and dilated, and was felt to contain still a large core of very hard fæces. The bowel was separated by blunt dissection from the vagina in front, and finally the opening into the vagina was encircled and the bowel was thus entirely freed at the lower end. An incision over the anal dimple was made and stretched out, the sphincteric muscle being felt all round it. The musculature of the lower end of the bowel was pushed up by blunt dissection, and the mucous and submucous coats were pulled through the new anus and stitched. The posterior incision was stitched up and drained, and the gap in the perineum was closed by stitches very similar to those used in a perineorrhaphy. No great shock was noticed, and the wounds healed in good time.

Further procedures centred round the removal of the hard fæces, which on several occasions required breaking up under anæsthesia. The new anal canal tended to be narrow and was dilated several times to size 18 Hegar. From early days, however, a degree of continence was noticed. Irrigation fluid could be held and discharged on instruction. Three months from the first operation the spur was crushed, and a week

[1] Accepted for publication on June 25, 1941.

after that the ends of the colostomy openings were turned in, and healing occurred almost at once.

Four months from the first operation finds the patient in much better general health, the colostomy closed, the vaginal defect soundly repaired, and an anus with tolerable control. There are occasional "accidents", but cooperation is present and full continence is expected. There is a tendency to narrowing of the anal canal, and the parents have been given a 17 and 18 Hegar dilator for daily use for a period.

In *Surgery, Gynecology and Obstetrics*, Volume lxxi, July, 1940, page 89, I found a very helpful account of a similar case by R. Franklin Carter and David Lyall of New York.

TWO RECENT CASES OF SLIPPED FEMORAL EPIPHYSIS TREATED BY PIN TRACTION.[1]

By H. K. CHRISTIE AND B. T. HOOPER,

From the Orthopædic Department, Wanganui Hospital, New Zealand.

A CASE of slipped femoral epiphysis reported by E. E. Dunlop in the January, 1940, number of THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY prompts us to report two recent cases treated on similar lines. The photographs of Dr. Dunlop's patient in fact show some slight loss of abduction and flexion; but internal rotation is not illustrated. It is this last movement that has been lost in our cases.

The clinical progress records show certain difficulties and pitfalls, and the end-result of the same method has not been equally good in the two cases.

A record of these cases after a further ten years would be most instructive.

Case I.

L.P.H., aged fourteen years, a schoolboy, complained of pain in the right hip one month prior to his admission to hospital.

On April 26, 1939, during a Rugby football match he was fallen on by the scrum. He finished the game, and did not notice pain in the right hip until he was sitting

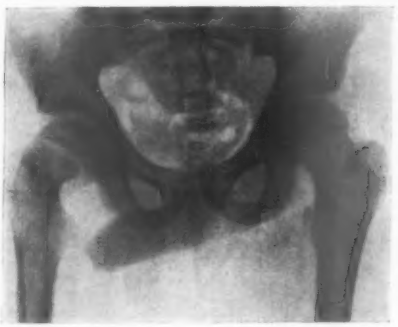


FIGURE I. Case I. Radiograph on admission to hospital. There is slipping and backward rotation of the right femoral epiphysis in relation to the femoral neck, together with upward displacement of the shaft, and a disturbance of Shenton's line. Rarefaction is taking place in the juxta-epiphyseal portion of the neck with increased width of the epiphyseal line.

down and had cooled off. It was especially painful when he tried to rise from the sitting position.

The pain now disappeared for seven days, but returned. A radiograph was taken, and showed no abnormality. Once more the pain eased off for a week, only to return more severely than before, and he was admitted to hospital.

Examination on his admission to hospital on May 25, 1939, showed that he was a normal healthy type of boy. He was unable to raise the right lower limb more than six inches from the bed. Passive flexion and abduction were limited to 30° and 40° respectively. Internal rotation was lost, but external rotation was increased almost to 90°. There was no tenderness on palpation over the joint, or on percussion of the heel.

X-ray examination on May 25, 1939, revealed a slipped femoral epiphysis on the right side, the result of a probable metaphyseal fracture, with consequent softening and decalcification of the adjacent diaphysis (see Figure I).

[1] Accepted for publication on April 5, 1940.

FIGURE II. Case I. Sound side.

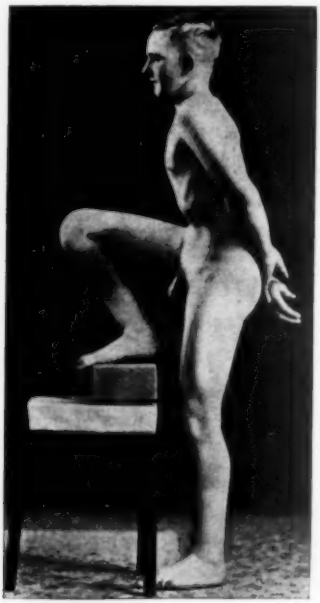


FIGURE III. Case I. Affected side.

Flexion is good, but is moderately reduced on the affected side.

FIGURE IV. Case I. Sound side.

FIGURE V. Case I. Affected side.

Internal rotation at the hip is almost completely lost.

On May 26, 1939, the hip was manipulated in full abduction (Whitman) and a plaster spica was applied. Control X-ray examination on May 30, 1939, showed that further slipping had taken place.

On June 5, 1939, a Kirschner wire was passed through the tibial tuberosity, and a weight of twenty-one pounds was applied. The usual fracture boards, elevation of the foot of the bed and traction of the other limb were used, both limbs being in Thomas splints. The affected limb was in adduction and internal rotation.

In three days reduction was apparently complete on X-ray examination, and the weight was reduced to ten pounds for ten days. Some relapse was then noted in the skiagrams, and the weight was increased to twenty pounds for two days, and then decreased to fifteen pounds for fourteen days, so that on July 6, 1939, good alignment was reported (one month's traction through the tibial tuberosity).

FIGURE VI. Case I. Abduction is moderately reduced, and some laxity of internal lateral ligament exists (pin traction through the tibial tubercle).

FIGURE VII. Case I. External rotation is increased on the affected side, and reaches 90°.

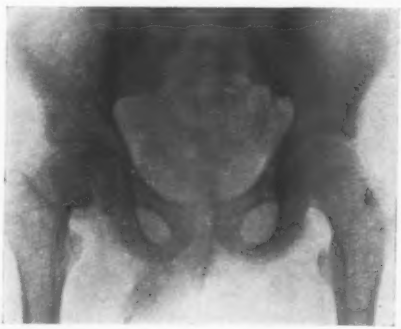


FIGURE VIII. Case I. Radiograph four months after admission to hospital. The relationship of the epiphysis to the neck is good, apart from some rotation, but the neck is now shortened and widened, with an irregularity of its juxta-metaphyseal margin. Local rarefaction is replaced by an apparent increase in density, but general rarefaction is present from immobilization.

FIGURE IX. Case I. Radiograph nine months after admission to hospital. A well marked *coxa vara* deformity is present and the epiphysis is splayed over the shortened neck. Normal density has returned in the neck, but small cystic areas of rarefaction still persist and the epiphyseal junction is still widened and irregular.

The knee joint had now become painful, and separation of the articular surfaces had occurred; the weight was reduced to ten pounds on July 7, 1939, and to five pounds on July 12, 1939. The knee was now very painful, and on July 13, 1939, the pin was removed and strapping extension to the thigh was substituted. The knee was immobilized with plaster slabs after a fitting had been made for a walking calliper splint.

On July 24, 1939, the patient was allowed up with the calliper (seven weeks' traction). X-ray examination showed that there was half an inch of displacement of the femoral epiphysis. The use of the calliper and other treatment were continued until February 14, 1940 (seven months from reduction).

On February 24, 1940, the patient had a normal gait, but three-quarters of an inch of shortening of the femur (*coxa vara*). The photographs show that internal rotation is lost (it was lost on the patient's admission to hospital and has not recovered), flexion is a little reduced, but the remaining movements are good (see Figures II, III, IV, V, VI and VII). External rotation was increased to 90°. In the skiagrams the femoral neck is seen to be shortened, which is responsible for most of the shortening of the limb (see Figures VIII and IX).

Case II.

O.E., aged fifteen and a half years, a schoolboy, was admitted to hospital with a note from his own doctor stating that three weeks previously he fell on the kitchen floor through slipping and his feet sliding from under him. He was unable to rise, was placed on a stretcher and had not walked since the accident. The provisional diagnosis made at the time of the accident was that of an impacted fracture of the neck of the femur.

On admission to hospital on August 21, 1939, he was seen to be a healthy boy

FIGURE X. Case II. Radiograph on admission to hospital. Marked slipping and backward rotation of the left femoral epiphysis have taken place with an upward and outward displacement of the upper end of the femur. Subchondral decalcification of the femoral neck is present.

FIGURE XI. Case II. Radiograph two months after admission to hospital. Good replacement of the epiphysis has been effected. Rarefaction is present along the juxta-metaphyseal portion of the neck, but is not so marked as in Case I, and Shenton's line is fairly well preserved.

of heavy but "doughy" type. The left leg was in the externally rotated position. There was some fullness in the left gluteal region. Measurement from the anterior superior iliac spine to the tip of the internal malleolus showed half an inch of shortening. There was limitation of all movements at the left hip.

All organs were healthy, although the pulmonary second sound was reduplicated.

Skiagrams taken on August 8, 1939, showed a separation of the left femoral epiphysis, with displacement of the neck and shaft upwards, and rotation of the shaft in relation to the head (see Figure X). These appearances were present in the film taken at the time of the accident by his own doctor, but were thought by him to indicate an impacted fracture of the neck. Thus in contrast with Case I there was in this case an immediate separation at the time of the accident.

On August 25, 1939, a Kirschner wire was passed through the lower end of the femur, and twenty-five pounds extension was applied as in the other case. The thigh

FIGURE XII. Case II. Sound side.

FIGURE XIII. Case II. Affected side.

Flexion is reduced in the affected hip. There is no lack of sexual development.

FIGURE XIV. Case II. Sound side.

FIGURE XV. Case II. Affected side.

Internal rotation at the affected hip is almost entirely lost.

was slightly abducted and the knee flexed to 30° to avoid external rotation. After ten days the X-ray examination showed that the epiphysis was in correct relation to the neck, but the head was subluxated downwards over the acetabular margin. Weight

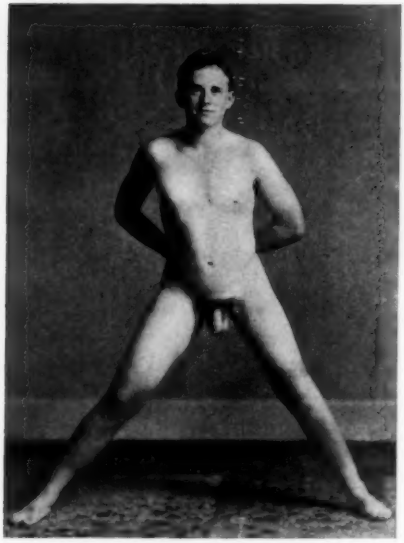


FIGURE XVI. Case II. Abduction.

FIGURE XVII. Case II. External rotation.

Abduction and external rotation are fully retained.

was then removed for three days, whereupon the dislocation disappeared, but the epiphysis again "slipped". We were back to the original position. A weight of ten pounds was then applied, but in three days it failed to effect reduction. The weight was increased to fifteen pounds, and after a further two days the X-ray report stated that there was now only slight upward displacement of the femoral neck. This weight was maintained for eighteen days with no change. The weight was then increased to twenty pounds with more internal rotation for a further three weeks.

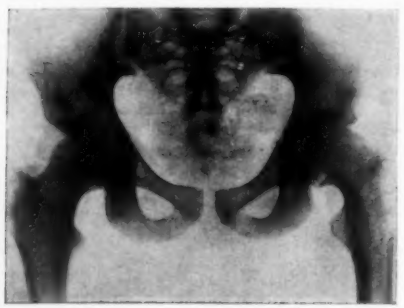


FIGURE XVIII. Radiograph six months after admission to hospital. The neck of the femur is widened, with splaying of the edges of the epiphysis. Normal bone density has returned except in the region of Babcock's triangle. A definite *coxa vara* deformity is present from the shortening of the femoral neck.

The X-ray report of October 20, 1939, after eight weeks' extension (see Figure XI), stated: "The epiphysis is in a relatively good position. General osteoporosis is present in this region, with decalcification a little more intense and localized in the region of the upper part of the metaphysis for the head and neck. As this is a lesion of the cartilaginous junction one cannot say to what extent union is taking place."

The wire was now removed, and the patient remained in bed until November 19, 1939, when he was allowed up to walk with a calliper.

On February 24, 1940, after six months (see Figures XII, XIII, XIV, XV, XVI and XVII), shortening is nil. Movements show that flexion is possible to 90° only, whilst internal rotation is lost. Abduction and external rotation are nearly normal. The film (Figure XVIII) shows satisfactory reduction of the epiphysis with no appreciable shortening.

Summary of Both Cases.

Both epiphyseal separations occurred at the fourteen to fifteen year period in healthy boys showing no evidence of hyposexualism.

In one case the separation occurred immediately on the fall, and was mistaken for a fractured femoral neck; in the other the separation was not apparent until three weeks after the accident and must have taken place gradually.

In both cases the condition on the patient's admission to hospital was one of shortening and external rotation with pain. Immobilization in plaster of Paris did not control the condition in the case in which it was tried.

Pin traction through the tibial tuberosity with the considerable weights involved led to stretching of knee ligaments, and the lower end of the femur was preferred for this reason.

The optimum traction would appear to be about twenty pounds for the first few days, followed by about fifteen pounds to maintain correction. In one case, however, twenty pounds actually caused dislocation of the hip joint. Traction is required for seven or eight weeks. There is then the alternative of a walking calliper or a patten and crutches.

In the result after six to nine months both patients showed considerable loss of internal rotation of the hip, with only slight loss of the other movements. The result shows that external rotation may actually be increased. *Coxa vara* resulted in one case from absorption of the neck without redisplacement.

Both these patients were healthy boys, and neither had a rise of temperature or pulse, or gave any other sign of infection.

BENIGN PAPILLOMA OF THE URETER.[1]

By G. H. BURNELL,
Adelaide.

PRIMARY tumours of the ureter, both benign and malignant, are comparatively rare, less than one hundred and fifty having been reported in the literature. The only cases known to me to have been reported from Australia are two which were reported by Bridge and Kirkland in 1934. Of the cases reported, more than two-thirds were malignant, so it will be seen that a benign primary ureteric tumour is quite an infrequent occurrence; it is for this reason that I report the following case.

A.E., a male patient, aged fifty-two years, was admitted to the Royal Adelaide Hospital on September 4, 1939. The following history was obtained. Eighteen months prior to his admission to hospital he had had an attack of pain in the left loin, radiating to the left subcostal region and to the left testis. He had not vomited, nor had there been any frequency of urination or dysuria; the pain had been relieved by morphine injection. For the previous twelve months he had been conscious of a dull ache beneath the left costal margin one hour after meals. Forty-eight hours before admission he had been seized with severe pain in the same situation as before, accompanied by an intense desire to defæcate and urinate; he had felt sick, but had not vomited; the pain had again been relieved by morphine injection.

On examination, nothing of note was found except that he had definite tenderness and rigidity in the left costo-vertebral angle; his temperature was normal; the urine contained no red cells or organisms.

On September 9, 1939, a urological investigation was made. A definite enlargement of the "middle lobe" of the prostate was found. While watching the urinary outflow from the ureteric orifices, one noticed that a papilloma protruded from the orifice on the left side with each peristaltic wave, retracting up the ureter and out of sight as soon as peristalsis ceased. It was obvious that the patient had a papilloma of the ureter.

The next problem was to decide whether the papilloma was secondary to others in the renal pelvis or primary in the ureter. For this purpose an attempt was made to pass a catheter up the left ureter, but the catheter would not pass more than five centimetres; at this point it was firmly obstructed, so the attempt to do a retrograde pyelogram was abandoned. A few days later an excretion pyelogram was prepared, the report on which was as follows:

"The left kidney outline is a little enlarged, and there is a dense shadow in the left side of the bony pelvis, caused by a calculus in the lower end of the ureter; there is a hydronephrotic dilation of the left kidney, due to the calculus."

An inspection of the plates gave no evidence of filling defects in the renal pelvis, such as one would expect if papillomata were present.

The question then arose as to the best method of treatment. It appeared probable that the tumour was a primary ureteric one, but the filling of the renal pelvis in the excretion pyelogram was not so good as is seen with the retrograde pyelogram, and this left a slight element of doubt as to whether papillomata might not be present in the renal pelvis as well. In view of the damage already done to the kidney, it was decided to remove the kidney, together with the whole of the ureter, including the ureteric orifice, the latter precaution being taken in view of the well-known tendency for renal papillomata to recur in the stump of the ureter, if the latter is not completely removed.

The operation was done in two stages on account of the patient's poor condition at the end of the first stage, which consisted of the removal of the left kidney together with about four inches of the upper end of the ureter. This was done on October 4, 1939. On October 18, 1939, through an oblique incision, an extraperitoneal approach was made to the left ureter, and this was isolated and freed down to the bladder. The bladder was opened, and a circular incision was made around the left ureteric orifice with an endothermy knife; the ureter was then stripped out and removed. Convalescence was uneventful.

The specimens, when examined, showed a simple hydronephrosis of the left kidney, with no evidence of papilloma in the renal pelvis or calyces. The condition found in the ureter is well illustrated in the colour plate (Figure I). It will be seen that there was an ovoid calculus lodged in the ureter six centimetres from the lower end. This calculus measured one centimetre in length and half a centimetre in diameter

[1] Accepted for publication on September 4, 1940.

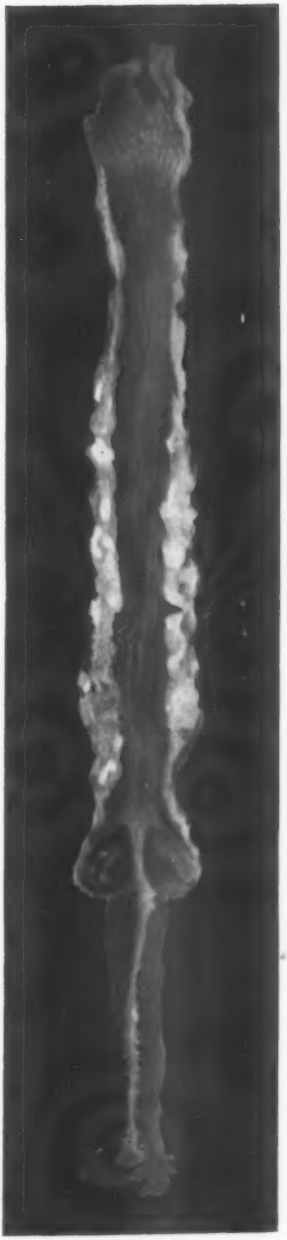


FIGURE I.

FIGURE II.

at its widest part. It had evidently been in that position for a long time, as it had formed a definite pouch in the ureter at that point. Arising from the wall of the ureter, just proximal to the pouch, was a slender stalk, which when followed was found to be six centimetres long and so just reached to the ureteric orifice. The first couple of centimetres of the stalk were quite smooth, but below that point it was covered with very short, fine fronds, while at its lower end there was a bulbous tip, consisting of compressed fine fronds (Figure II).

Sections of the growth were examined by Dr. R. C. Bassett. He reports that the tumour is undoubtedly a benign papilloma. Unfortunately, owing to the delay in having the sections cut while the specimen was being reproduced in colour, a certain amount of spoiling took place, with the result that the microscopic sections are not good enough for purposes of reproduction.

It is interesting to speculate on the ætiological relationship of the calculus to the tumour, if any. One feels that if the tumour formed as a result of the irritation of the calculus, then such tumours should be much commoner than they are, as obstruction by a ureteric calculus is a common condition. On the other hand, many ureteric growths have been reported unassociated with calculus. It will be noticed that the stalk of the papilloma arises from the wall of the ureter proximal to the calculus; but this does not preclude the idea that the presence of the papilloma caused the passage of the calculus to be obstructed, as it is possible that when the calculus first descended the ureter the papilloma was more compact than it was found to be at operation, and was sufficiently large to hold up the calculus when the latter passed the stalk and came in contact with the main mass of the papilloma. If this is so, then it must be that the papilloma has subsequently become elongated by the peristaltic action of the ureter.

Acknowledgement.

My thanks are due to Mrs. A. Bateman for her excellent reproduction of the appearance of the specimen.

HÆMANGIOMA OF THE HAND, INVOLVING PHALANGEAL BONES, WITH DISTINCTIVE RADIOLOGICAL APPEARANCE.[1]

By KONRAD HIRSCHFELD,
Brisbane.

OWING to the special functions of the hands and feet, hæmangiomata occurring in these situations present problems which do not arise in their treatment elsewhere, and the following case is of interest on this account.

FIGURE I. Lateral view of hand.

L.Y., aged twenty-four years, complained of pain and of swelling in his left hand. He sought advice not only for these reasons, but also because he had been advised to refrain from working as the bones of his hand were diseased.

For fifteen years he had had swelling of his left hand, chiefly of the distal part of the palm and of the index and middle fingers. His hand ached occasionally, particularly in hot weather when the swelling increased in size. Ten years ago he had received treatment with an "electric needle". Three years later he had further treatment of the same kind. Two months ago he had ultra-violet light treatment and his hand was examined by X rays. This revealed rarefaction of the phalanges, and the patient was advised to cease work lest he should sustain a fracture of the bones involved.

Recently he had had pain in his hand and in the middle finger, in addition to the aching previously mentioned.

On examination, on the radial and distal aspects of the palmar and dorsal surfaces of his left hand there was a soft elastic swelling which extended slightly onto the palmar and considerably onto the dorsal aspects of his index and middle fingers. As this swelling was traced onto the fingers the outline of large tortuous veins could be seen. The swelling was less towards the terminal phalanges, but through the nails of both index and middle fingers a blue colour was seen which faded on pressure. The swelling on the hand could be felt as a globular mass between the heads of the second and third metacarpals, and the knuckles of the corresponding fingers were separated by it. The skin over the palmar aspect of the mass was stippled with small white scars, evidently from the previous treatment. The swelling increased in size when the arm was dependent and the patient complained that his fingers ached in this position. There were no abnormalities to be observed in the radial or ulnar vessels at the wrist, nor were any large vessels to be found leading to the tumour.

A radiograph revealed a very distinctive picture. There was marked separation of the distal ends of the second and third metacarpals. There was slight thickening

[1] Accepted for publication on March 13, 1941.

in the periosteum of the metacarpal bones, with some areas of rarefaction in the cortex. The proximal phalanx of the index finger showed a central area of rarefaction as if several rounded areas had become confluent. The other phalanges of this finger were normal. The proximal phalanx of the middle finger was slightly wider than that of the index finger. There was generalized, though slight, rarefaction of the central portion of the bone with a well marked rounded rarefaction towards the distal end. The second phalanx showed similar but more clearly defined changes, while the terminal phalanx was normal, as were the other bones of the hand. There was one small opacity in the soft tissues opposite the head of the third metacarpal. These appearances are consistent with the changes produced in bones by external irritation.

FIGURE II. Palmar view of hand. The small white spots seen on the palm of the hand are the diathermy scars. Note the dilated veins, particularly on the middle fingers.

A diagnosis of plexiform hæmangioma of the hand was made. As the patient wished to be relieved of his symptoms and also to return to work, treatment was attempted by injection of sodium morrhuate; fifteen cubic centimetres of a 5% solution of sodium morrhuate were injected in various sites into the swelling. The injection was made by inserting the needle into the swelling and, after blood was withdrawn into the syringe, the solution was injected.

Following the treatment there was a severe reaction. The patient developed gross œdema of his hand and fingers and œdema of the forearm extending almost to the elbow. He complained of severe pain in his hand, and had fever up to 100° F. Six days after the injection, while the swelling was at its maximum, he developed acute tonsillitis and his temperature rose to 103·5° F. This settled down with conservative treatment, and fourteen days after the injection his temperature was normal. By this

FIGURE III. Lateral skiagram of hand.

FIGURE IV. Antero-posterior skiagram of hand.

time the swelling of the patient's hand and forearm had almost disappeared, but the skin over the injected area was peeling. Movements in the two fingers concerned were slightly impaired.

On discharge from hospital one week later the patient had full movements of all his fingers. His pain had disappeared, the tumour had become hard, and there was no change in size when the hand was dependent. Nine months later there were no signs of patency of the tumour. The patient had not noticed any pain ncr aching in his hand or fingers and he had been working. The radiographic appearances were unchanged.

The case is of interest because of the unusual situation and because of the bone changes which present such a distinctive radiographic picture.

The radiological appearances of hæmangiomata which have been described are of two kinds. The first are opacities in the soft tissues due to phleboliths in the dilated vessels; the second are changes due to involvement of bone. The bone in such cases shows areas of rarefaction which are arranged so as to present a honeycombed appearance, and these changes have so far been described chiefly in the flat bones and in the vertebræ.

Acknowledgements.

I wish to thank my former chief, Mr. Alan Perry, of the London Hospital, for his kind permission to publish this case, and for his advice and help, and Dr. Jupe for his help in interpreting the radiograms.

AN ANOMALOUS ANASTOMOSIS BETWEEN THE INTERNAL CAROTID AND BASILAR ARTERIES.[1]

By SYDNEY SUNDERLAND,
Anatomy Department, University of Melbourne.

THE anomalous anastomosis between the internal carotid and basilar arteries, here described, was observed in an adult white male dissecting-room subject, aged eighty-eight years, and presented such unusual features that it was thought worthy of record. The comparative rarity of the condition and the close anatomical relationship of the anastomosing vessel to the sensory root of the fifth cranial nerve are features which, it is believed, merit attention.

FIGURE I. Photograph taken through the *incisura tentorii cerebelli*, the left margin of which has been divided and displaced, to show the course taken by the anomalous vessel in the posterior cranial fossa. The terminal portion of the basilar artery is seen in the centre of the field and posterior to it the divided surface of the pons, which is partly obscured by the curved right margin of the incisura. The relationship of the anomalous vessel to the sensory root of the trigeminal nerve, as these structures cross the apex of the petrous temporal to enter Meckel's cave, is well shown.

The Anomaly.

The anatomical features of the anomaly are well illustrated in Figure I. The anastomotic vessel, which was present on the left side, branched from the basilar artery about midway up the pons at the level of entrance of the fifth nerve roots. It coursed laterally over the abducent nerve till it reached a position posterior to the upper margin of the apex of the petrous temporal and immediately medial to the sensory root of the trigeminal nerve. The vessel then curved forwards and accompanied the nerve through the trigeminal foramen into Meckel's cave. As the nerve and artery crossed the apex of the petrous temporal, the latter was in direct contact with the flattened upper surface of the sensory root over its inner aspect, which it slightly grooved. In Meckel's cave the vessel arched downwards, internal to the Gasserian ganglion. It then turned medially, pierced the inner dural wall of the cave and entered the cavernous sinus, where it joined the lateral aspect of the internal carotid artery just after that vessel had entered the sinus.

The anastomotic vessel was unusually large, being twice the diameter of either posterior cerebral artery and a little over half the diameter of the basilar artery, which was of normal dimensions. The remaining branches of the basilar and the formation and branches of the circle of Willis were all normal. On the same side, however, the vertebral artery was very small, being only

[1] Accepted for publication on February 21, 1941.

one-fourth the size of its fellow of the opposite side. The vessels showed no pathological changes.

Discussion.

Anomalous anastomoses between the internal carotid and basilar arteries have been reported by Quain (1844), Tüngel (1860), Duret (two) (1874), Tarenieckí (1880), Hochstetter (two) (1885), Flesch (1882), Decker (1886), Batujeff (1889), Elliot Smith (1909) and Oertel (1922). That described by Batujeff in no way resembles the other anomalies. In his case the basilar artery took origin from the left internal carotid artery at the base of the skull and entered the posterior cranial fossa through the anterior condyloid foramen, while in each of the remaining cases the aberrant vessel branched from the internal carotid artery in the cavernous sinus and passed posteriorly either through (Quain, Tüngel, Tarenieckí, Hochstetter (two), Oertel) or around (Duret (two), Flesch, Decker, Elliot Smith) the *dorsum sellæ* to reach the basilar artery. The condition was present on the right side in the cases described by Hochstetter (one), Tarenieckí, Tüngel, Duret (two) and Oertel, while in the remaining cases the anomaly was observed on the left. In the cases described by Elliot Smith, Decker and Flesch the anastomotic vessel was related, while in the cavernous sinus, to the Gasserian ganglion and its branches. In these cases, however, the vessel ran a slightly different course to reach the basilar artery from that recorded in this paper, and from their accounts it apparently did not bear the same intimate relationship to the sensory root of the fifth nerve. In all three cases the vessel was separated from the sensory root by a dural band as the former skirted the lateral margin of the *dorsum sellæ* to enter the posterior cranial fossa.

The manner in which such anastomoses become established is readily explained. It is well known that in the embryo many small anastomotic channels linking the internal carotid and vertebral arterial systems develop at the base of the brain. In older embryos these become reduced to the posterior cerebral arteries, which subsequently transfer their origin from the internal carotids to the basilar artery which has been previously outlined by the fusion of the cranial extremities of the vertebral vessels. As a relic of this embryonic relationship, it is not unusual to see the posterior cerebral artery taking origin from the internal carotid after that vessel has emerged from the cavernous sinus. The anomaly under discussion, however, represents the persistence of one of the early anastomotic channels which are usually absorbed as development proceeds, and this fœtal channel may quite possibly have been retained as a compensatory mechanism to offset the small vertebral artery on that side.

The interest and importance of this vascular anomaly is the close relationship which the anastomosing vessel bears to the sensory root and Gasserian ganglion of the trigeminal nerve—a relationship so intimate as to leave an impression on the nerve root. It has been suggested by Dandy (1939) that pressure on the sensory root by a branch of the basilar artery is responsible, in the majority of cases, for the pain in *tic douloureux*. In this particular case there can be no question concerning the pressure exerted on the nerve root by the vessel—a pressure, moreover, which must certainly have been greatly increased by the pulsations occurring in a vessel of the size present. Yet the past history in this case when investigated contained no record of any disturbance of fifth nerve function.

Acknowledgement.

I wish to thank Mr. L. Preston for the photograph of the specimen accompanying this paper.

Bibliography.

N. Batujeff: "*Eine seltene Arterienanomalie (Ursprung der A. basilaris aus der A. carotis interna*", *Anatomischer Anzeiger*, Volume iv, 1889, page 282.

Walter E. Dandy: "Lesions of the Cranial Nerves", *The Journal of the International College of Surgeons*, Volume ii, January, 1939, page 5.

F. Decker: "*Über eine seltene Varietät der Arterien der Hirnbasis*", *Sitzungsberichte der physikalischmedizinischen Gesellschaft zu Würzburg*, 1886; quoted by Oertel, *loco citato*.

H. Duret: "*Recherches anatomiques sur la circulation de l'encéphale*", *Archives de physiologie normale et pathologique*, 1874, Volume i, Second Series; quoted by Oertel, *loco citato*.

M. Flesch: "*Untersuchungen über Verbrechergehirne*", Würzburg, 1882, page 52; quoted by Oertel, *loco citato*.

F. Hochstetter: "*Über zwei Fälle einer seltenen Varietät der A. carotis interna*", *Archiv für Anatomie und Entwicklungsgeschichte*, 1885; quoted by Oertel, *loco citato*.

Keibel and Mall: "Manual of Human Embryology", 1912, Volume ii, page 498.

Franklin P. Mall: "On the Development of the Blood-Vessels of the Brain in the Human Embryo", *American Journal of Anatomy*, Volume iv, 1905, page 1.

Oertel: "*Über die Persistenz embryonaler Verbindungen zwischen der A. carotis interna und der A. vertebralis cerebralis*", *Anatomischer Anzeiger*, Supplement to Volume lv, 1922, page 281.

R. Quain: "The Anatomy of the Arteries of the Human Body", 1844, page 507.

G. Elliot Smith: "Note on an Anomalous Anastomosis between the Internal Carotid and Basilar Arteries", *Journal of Anatomy and Physiology*, Volume xliii, 1909, page 310.

Tareniecki: *Militärärztliches Journal, Juniheft*, 1880 (*russisch*); quoted by Oertel, *loco citato*.

Tüngel: "*Klinische Mitteilungen des allgemeinen Krankenhauses zu Hamburg*. 1860", 1862, page 258; quoted by Oertel, *loco citato*.

Reviews.

Fractures and Other Bone and Joint Injuries. By R. WATSON-JONES, B.Sc., M.Ch.Orth., F.R.C.S.; Second Edition, 1941. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 736, with 1,040 illustrations. Price: 50s. net.

As might be expected in a new edition appearing so soon after the first, the general treatment of this standard work follows closely on that in the first. Chapter x, on open and infected fractures, has been brought up to date in the light of war experience under the title of "Open Fractures and War Wounds", with an increase of three pages. A little juggling with the numbering of blank pages immediately restores the correspondence so that the number of pages is the same in the two editions. Of the general statements in this chapter there can be little criticism. There are some interesting remarks on amputations.

The list of authors quoted has been revised and materially increased, which is one reason among many why the book should appeal to specialists and practitioners alike.

The Art of Surgery: A Text-Book for Students and Practitioners. By H. S. SOUTTAR, D.M., M.Ch., F.R.C.S.; Fourth Edition, in two volumes; 1940. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 789, with illustrations. Price: 25s. net.

THIS edition, the fourth in ten years, appears for the first time in two handy volumes. This innovation will appeal to those students who like to refer to their textbook during duty in the surgical wards.

Mr. Souttar's object has been to describe very fully what is fundamental, and to omit what is not essential, and he claims that the book is now fully up to date. He has attained his object, and has made good his claim. Any student who has absorbed the contents of "The Art of Surgery" should have no difficulty in passing an examination in the subject. The author believes in the use of the visual memory, and to that end has continued the use of the thumb-nail sketches which were a feature of the earlier editions. The tendency in modern surgical textbooks to instil knowledge via the visual memory is doubtless due to the cinema, and is likely to become still more pronounced.

As one would expect from a surgeon who has had great experience in the teaching of general surgery, a nice balance has been preserved in dealing with the various subjects and regions. The final two chapters of the first volume on "Injuries and Infections of the Brain" and "Tumours of the Brain" are admirably concise, and contain just what a student ought to know of the subjects that are tending to drift entirely into the hands of specialists. We have tested the up-to-dateness of the work in several directions. Two only need be mentioned—a place has been found for sulphonamide therapy, and Leriche's recent work on the mechanism of the production and on the treatment of acute traumatic synovitis is referred to. Not every method of treatment advocated by the author will gain general acceptance—for example, thoracoplasty or drainage by operation of the dilated bronchial tubes in bronchiectasis, or Phelps's open incision in the treatment of *talipes equino-varus*.

Variety, however, is the spice of life, and these criticisms do not detract from the value of the book.

In conclusion it may be said that the style in which the work has been written has made it a pleasure to read—a merit not possessed by every textbook of surgery. The price is unusually reasonable.

Surgery of Modern Warfare. Edited by H. BAILEY, F.R.C.S.; Part IV; 1941. Edinburgh: E. and S. Livingstone. Super royal 8vo, pp. 160, with 177 illustrations. Price: 12s. 6d. net.

IN this volume the section on wounds of bones and joints is concluded. Especially valuable is the chapter on general principles of amputations by Sir William de Courcy-Wheeler, and that on amputations from the point of view of fitting artificial limbs by Jenner Verrall, wherein he defends the use of the guillotine amputation under certain conditions.

The rest of the volume is devoted to wounds of the face and neck, including those of the eye and ear. Kilner's contribution on wounds of the face and jaws is essentially a chapter on reconstructive surgery. This work is not for the general surgeon, and should never be attempted by the surgeon who is "trained but not necessarily highly experienced", but for whom, as the advertisement on the back cover states,

this book is written. In the same way it seems doubtful whether some of the surgery described in the chapters on injuries to the eye and orbit should be undertaken by other than specialists in this branch of surgery. The urgent treatment of certain conditions must of course fall to the lot of the medical officer who first sees the patient, but most of it can wait until the trained man is available.

Injuries of the Skull, Brain and Spinal Cord: Neuro-Psychiatric, Surgical and Medico-Legal Aspects. Edited by SAMUEL BROCK; 1940. London: Baillière, Tindall and Cox. Crown 4to, pp. 649, with 63 illustrations. Price: 38s. 6d.

THIS book is the work of twenty distinguished collaborators, chiefly American, under the editorship of Samuel Brock. Brock asserts in the preface that it is "a series of comprehensive monographs on neurology, psychiatry, neurosurgery (in its civil and military aspects), radiology and forensic medicine as far as these subjects relate to injuries of the skull, brain and spinal cord. Moreover, obstetrics and pediatrics are represented in the fields of birth injury." This fairly describes the work.

The mood is dogmatic where possible and necessary, yet shows a nice balance on matters still controversial. This is well shown in the chapters "Fracture of the Skull" and "The Relationship of Birth Injury to the Other Organic Diseases of the Brain".

It has not been possible to distinguish by chapter the surgical, medical and legal aspects of injury; this has been attempted, however, in a general way.

A brief consideration of the general pathology of brain injury is followed by several monographs which deal with the surgical aspect of trauma. Even here the subjects of contusion and laceration have been entrusted to an English physician who has made notable contributions to our understanding of the symptoms and sequelæ of these disorders. Cerebral birth injury, post-traumatic convulsive states, neuroses and psychotic states are given full treatment. Useful chapters, more particularly related to medico-legal affairs, are those on "Stimulation" and "The Relationship of Brain Injury to Other Organic Diseases of the Brain". The section on the spinal cord and spine has the same general arrangement and occupies about one-sixth of the book. A valuable chapter on the effects of electric shock, caisson disease, cerebral fat embolism and cerebral air embolism precedes a concise but comprehensive account of strictly medico-legal matters, which closes the work.

The book brings to the reader much rich experience and knowledge, of which the latter may be fortified by the useful bibliographic references to be found at the end of each chapter. Physicians, surgeons and lawyers will find it most useful.

A Complete Outline of Fractures (Including Fractures of the Skull) for Students and Practitioners. By J. G. BONNIN, M.B., B.S., F.R.C.S., M.R.C.O.G.; 1941. London: William Heinemann (Medical Books) Limited. Medium 8vo, pp. 519, with 575 illustrations. Price: 25s. net.

THE author describes the usefulness of his work in the introduction: "This book is my ideal students' textbook, written, I hope, before the pursuit of obscure points has blinded my mind to the simpler demands of the student. . . . In writing this book I have written the book I desired to find in my late student and early post-graduate days, and which, unhappily, was not to be found. It is written in the hope that many others may find in it the book they have sought, and that many more may find it a simple yet fairly complete exposition of fractures." The various subjects are disposed of in an orderly manner which blends the simplicity of description of diagnosis and types of fractures for the instruction of students with the more difficult account of technicalities for surgeons. Böhler's methods are followed largely, but Böhler has abandoned some of the methods described—for example, skeletal traction for fractures of both bones of the forearm and double pinning and incorporation of the pins in plaster in tibia and fibula fractures, especially walking in such a cast, as infection of the pin holes often follows weight bearing. Many familiar illustrations are not so well reproduced as when they originally appeared. This applies to most of the illustrations which the author acknowledges are "often in deliberate imitation of illustrations in other books". Nowadays anyone who wishes to break new ground in a book on fractures, unless he has a special message, has a difficult task before him. This book has the title "A Complete Outline of Fractures", but as there are 493 pages and 575 illustrations the book is more complete than an outline; it should therefore not be regarded as a students' compendium, but be compared with the well-known books on fractures. In the latter regard there has never before been such a high standard to which a new author must attain.